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Influence Of Some Domestic Environmental Factors On The Prevalence Of Respiratory Symptoms And Lung Function Changes In Residents Of Three North American Townships

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INFLUENCE OF SOME DOMESTIC ENVIRONMENTAL FACTORS ON THE PREVALENCE OF RESPIRATORY SYMPTOMS AND LUNG FUNCTION CHANGES IN RESIDENTS OF THREE NORTH AMERICAN TOWNSHIPS

by

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the requirement for the degree of
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ABSTRACT

All residents, aged 7 years and above, of three towns were canvassed to participate in a study to determine whether domestic air pollution factors were important in explaining respiratory diseases. Of the 7,203 who participated, this study covers only the 4,074 who were non-smokers. A standardized questionnaire was administered which determined the frequency of cough, phlegm, wheeze, dyspnea, and also domestic factors such as exposure to domestic pets, exposure to gases, vapours and dusts from hobbies, exposure to emissions from gas stoves, the use of fireplaces, air conditioners, humidifiers and heating systems, domestic crowding, and the number of smokers in the home. The forced expiratory volume in one second (FEV_1), maximum expiratory flow volume at 25% of vital capacity ($MF_{25\%}$) and maximum expiratory flow volume at 50% of vital capacity ($MF_{50\%}$) were obtained from lung function tests on all subjects.

A small group of symptomatic housewives (26) and matched controls (26) were selected for further study of their actual exposure to sulphur dioxide (SO_2), nitrogen dioxide (NO_2), and respirable suspended particulates (RSP) within the home. Levels of these pollutants were also determined outside the homes.

Domestic pets and the use of fireplaces and humidifiers had no consistent effects on the reporting of respiratory symptoms nor on lung function when considered individually or in combination with the other exposure factors. Hobbies which exposed residents to gases, vapours and

dust, the use of gas stoves, the absence of air conditioning, the use of hot water heating systems, crowded homes, and the presence of smokers in the home all had effects on reporting of symptoms and lung function. The greatest effect on lung function was observed with the use of hot water heating systems, whereas the lowest effect was observed with the number of smokers in the home. There was consistent interaction among the exposure variables by 2-way, 3-way, 4-way and 6-way comparisons. The FEV_1 of white children showed consistently the greatest responses to all of the exposure variables, so much so, in the 3-way interactions, white boys and girls who lived in homes which had the exposure factors had mean FEV_1's of between 0.274 to .397 liters lower than their counterparts who lived in homes which did not have the exposure factors. In a 4-way interaction comparison, white boys and girls who had hobbies which produced gases, vapours and dusts, and lived in homes without air conditioning, but with gas stoves, and with hot water heating systems had mean FEV_1 of .387 liters and .461 liters respectively lower than their counterparts who had no hobbies and lived in air conditioned homes with forced air heating and with electric stoves.

In black children and adults, the only variable that consistently was associated with increased reporting of symptoms and lung function depression was the use of hot water heating systems.

Indoor levels of NO_2 and RSP were always higher than the corresponding outdoor levels. The use of gas stoves resulted in about 100 ug/m^3 more NO_2 indoors than the use of electric stoves. The presence of one

smoker and 2 or more smokers in the home resulted in 8 ug/m^3 and 20 ug/m^3 RSP respectively greater than in homes with no smokers. NO_2 and RSP correlated separately with the lung function variables and there was no interaction. There were no significant differences in air pollution levels in the homes of cases and controls.

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I thank the people of the three towns for their cooperation in the study, especially the group of housewives who were very tolerant when the noise of the personal sampler turned out to be higher than the "hum from an air condition unit". The Department of Environmental Protection, State of Connecticut and the Department of Environment and Air Resources, State of South Carolina, were helpful in allowing me the use of their Laboratories for some of the analyses.

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CHAPTER 1

INTRODUCTION

In 1661, John Evelyn, author of "Smoake of London", wrote "the hellish and dismal cloud of 'sea coale' - impure and thick mist accompanied with a fuligenous and filthy vapour - corrupting the lungs - so that Catharrs, Phthisicks, Coughs and Consumption range more in this one city than in the whole earth besides".

For centuries, pollution of the air has been associated with respiratory diseases, but even now there is an incomplete understanding of the true causative factors and mechanisms whereby the tissues of the respiratory tract are affected to produce disease. Attempts have been made to assess these factors from provocation studies using various animal models, controlled studies using healthy human adults, and through epidemiological studies in communities and in occupational environments. Despite these attempts, numerous gaps in knowledge still exist.

1.1 Overview

The study of the health effects of certain air pollutants on the respiratory tract becomes more complex as we move from the experimental to the epidemiological, but at the same time, the utility with regards to determining the causal and consequently the protection factors also increases in the same direction.

Animal studies are useful in determining dose-response curves for a single pollutant by using different outcome modes such as biochemical changes, tissue changes, morbidity or mortality. In most animal studies, the doses used are orders of magnitude greater than those encountered in the ambient environment or work environment and therefore it is difficult to extrapolate the findings to man, either qualitatively or quantitatively. Species susceptibility, hyperactivity, and metabolic differences are also major limitations in using animal models for determining responses to respiratory irritants. Nevertheless, these experimental models are useful in determining pathology once the causal factors have been established.

Acute respiratory responses in normal humans at rest or during exercise or susceptible subjects have been studied under controlled conditions using single pollutants or simple combinations. These types of studies are useful in determining thresholds for acute responses to specific pollutants, but fall short in explaining the real situation where the exposures are for longer durations and the exposure patterns extremely complex. Usually in such determinations, the ethical considerations are paramount and therefore the degree and extent of exposure has to be controlled to ensure reversibility of any response. Because of these shortcomings, extrapolation to the general public, which is composed of a broad range of age, ethnic and susceptibility groups, is somewhat limited.

Epidemiologic studies provide the basis for determining risk factors in disease, but are sometimes difficult to interpret because of confounding

host and exposure factors. The host factors which have to be analyzed include age, sex, race, hypersusceptibility, pre-existing diseases, and familial aggregation; environmental exposure factors include pollution type i.e. whether the effect is on the lower or upper respiratory tract, meteorologic conditions, socioeconomic factors, migration for health or social reasons, occupational pollutants, domestic exposure, and smoking.

The inherent weaknesses in challenge studies using animals and volunteer subjects do not allow the opportunity to determine the true risk factors of community respiratory diseases. Epidemiological studies in the past focussed primarily on outdoor pollution as the major causal factor, despite the fact that most people spend about three quarters of their time within the home, and in fact only about 5% of their time outdoors (Szalai, 1972).

The methods for assessing exposure to pollutants have gradually evolved, and one major weakness in the past was the use of air pollution data, generated by government agencies, which were primarily designed for assessing air pollution emissions to the outdoors. No attempts were made to assess personal exposure; since this is the only valid way to determine whether air pollution was a major factor in respiratory disease causation. In some cases the outdoor exposure was estimated from coal consumption, and in cases where air pollution data were collected to assess population exposure, single sampling sites were used to assess exposure of large metropolitan areas.

A variety of questionnaires have been used to assess symptom prevalence, vital statistics and in some cases, smoking history. Up to the time of this study, no questionnaire was designed to incorporate questions on domestic factors.

Summary

Residents of many low pollution communities have respiratory diseases which cannot be explained in terms of medical history, smoking history, occupational exposures or previous residence in a high pollution district. Even in high pollution communities, the findings on respiratory diseases and lung function are inconsistent with the air pollution levels. Hence it is only prudent to examine what other factors may initiate and aggravate respiratory diseases. The domestic environment has its own generating sources because of numerous activities within the home, and there is evidence that pollutants may enter the home from the outside. Many of these pollutants are respiratory irritants and toxins, and in this study an attempt will be made to determine the nature of the exposure and the ways in which the respiratory health of residents in three American townships was affected.

1.2 Study Objectives

In order to determine whether the domestic environment was a risk factor in respiratory health, this study was divided into two parts:

i. a A standardized questionnaire was used on a group of residents from three townships to determine the prevalence of cough, phlegm, wheeze and dyspnea.

b To determine from this questionnaire the existence of nine potential domestic exposure factors.

c To measure the lung function of all respondents of the questionnaire.

d To use the symptom and lung function data to determine whether they are influenced by the following domestic factors: pets, hobbies which may expose residents to gases, vapour and dusts, heating fuel, cooking fuel, fireplaces, air conditioning, humidifiers, crowding, and the number of smokers in the home.

ii.a To develop a domestic air pollution sampler and to use it to measure exposure to SO_2 (sulfur dioxide), NO_2 (nitrogen dioxide) and RSP (repirable suspended particulates).

b To use the above sampler to assess the exposure characteristics of a group of housewives selected from the total population because of their exposures and work histories.

c To use the data base generated for the three pollutants to validate any of the findings from the questionnaire study (ie. above).

A critical review of the literature is given in chapter 2 to illustrate the importance of the domestic environment in respiratory disease causation.

CHAPTER 2

LITERATURE REVIEW

This review will show in Section 2.1 how the domestic environment is important with regards to the generation and containment of pollutants that may affect respiratory health. This Section is preceded by a short discussion and a table on the air quality standards for SO_2, NO_2, ozone(O_3), and total suspended particulates (TSP) since this information will serve as a reference for exposure data from the literature. In Section 2.2, the ways in which the individual pollutants affect the lung tissues will be covered; the emphasis being on challenge studies with animals and human subjects. In Section 2.3, the epidemiological studies will be reviewed, the emphasis being placed on the significance of the domestic environment in relation to the outdoor environment. This last section will also contain a brief review of the studies on children since this aspect is relevant to the study on domestic exposures.

2.1 Domestic Air Pollution Sources

2.1.1. Air Quality Standards

Most industrialized countries have developed air pollution standards to protect the health of the public. In the U.S.A., the first Federal attempt to conduct a program was in 1955, and in 1963 the Clean Air Act

was adopted. Further to this, the Air Quality Act was enacted in 1967 and one regulation for this Act was the development of air quality standards. Standards for SO_2, NO_2, TSP and O_3 were promulgated in 1971 (Federal Register, 1971), the limits being based mainly on the health of the total population. For each pollutant, literature evaluations were undertaken as to the effect of the pollutants on man, animals and vegetation (U.S. Department of Health, Education and Welfare, 1969 (SO_2); 1970 (O_3); 1969 (TSP); 1969 (NO_2)).

The response to a toxic material is firstly biochemical, which may not progress further to a physiologic response providing the dose is kept below the level required to produce such a response. In most cases, the biochemical response is reversible with no permanent damage. Increased insult may progress to the next step which is a measurable physiologic response whereby, removal of the provoking material will most likely result in a cessation of the response. There may not be any permanent damage and the response may be reversible providing the insult is not repeated. Repeated exposures may lead beyond the compensatory stages to disease or eventually to death. The air quality standards have been based on no-detectable physiologic response and apply only to the outdoor environment. Table 2.1 shows the standards for SO_2, NO_2, O_3 and total suspended particulates.

Table 2.1 - Air Quality Standards

Pollutant	Time	Air Quality Standards (ug/m^3) Time Weighted Average	ALERT+	WARNING+	EMERGENCY+	SIG. HARM+
SO_2	1 yr	80	800	1600	2100	2620
	24 hr	365	---	---	---	---
NO_2	1 yr	100	---	---	---	---
	24 hr	---	282	565	750	938
O_3	1 hr	160	200	800	1200	1400
TSP*	1 yr	75	375	625	875	1000

\+ = These are Connecticut levels that are declared whenever the concentration of one or more of the pollutants listed above, reaches the prescribed levels at any monitoring site and meteorological conditions are such that the pollutant concentrations can be expected to remain at the above levels or increase over a period of 12 hours or more. At each stage, numerous control strategies are adopted. (State of Connecticut, 1971).

TSP* Total suspended particulates. Some studies have reported respirable suspended particulates (RSP) which consist of particles less than 10 um in diameter; no outdoor standards exist for RSP.

2.1.2 Sources

Indoor air pollution can result from numerous sources. It can diffuse from the outside through windows, doors, ventilators, cracks and air intake systems. Activities such as working, heating, cleaning, smoking and hobbies can also result in gases, vapours and particulates becoming airborne within the home. In some cases degradation of the walls, furniture and floor coverings can also result in the production of airborne matter in the home. The keeping of pets within the home may produce exposure to hair, fur, ectoparasites, droppings and dusty foods.

Enclosures may provide some protection from outdoor pollution, but they may also entrap pollutants that may have infiltrated or have been generated indoors (Sterling et al, 1977 & 1982). Yocom et al (1971) indicated that diffusion can account for up to 50% of the indoor loading, especially for particulates or organic materials. They also stated that the domestic environment may have its own pollutant sources e.g. from stoves and fireplaces, but they did not try to determine what proportion of the total burden was from indoor generation. In buildings with a large percentage of make-up air, e.g. office towers and apartment complexes, diffusion resulted in indoor levels being about two thirds of the outside levels (Thompson, 1973). Andersen (1972) found a high correlation ($r=.52$) between indoor and outdoor SO_2 and an even higher correlation ($r=.83$) for particulate matter; the building studied had an air make-up system. Derham et al (1974) found similar correlations for NO_2 and CO, but found that it was closely related to the ventilation rate of the building.

Two studies which measured lead (Dienal et al, 1981; Halpern et al, 1978) also showed that diffusion from outside accounted for a large proportion of indoor lead.

The Harvard six city study also implicated diffusion as being important in indoor levels for particulate matter and sulphates (Dockery and Spengler, 1977; Dockery and Spengler, 1981 a&b; Spengler et al, 1981); the use of air conditioners was found to result in reduced infiltration.

Smoking, cleaning and dusting generated a large amount of dust within the home (Lefcoe et al, 1971). In another study, Lefcoe et al, (1975) found that indoor levels decreased during the night, probably because of lower outdoor levels and lower indoor activity. These researchers indicated that vacuum cleaning and bed-making resulted in considerable generation of particles greater than 1 um in diameter.

In homes with cigarette smokers, a large percentage of the aerosol matter came from the sidestream smoke and exhalation emissions (Harke, 1974). Other pollutants such as acrolein, acetaldehyde, nicotine and polycyclic aromatic hydrocarbons are emitted, some of which might be respiratory irritants. Binder et al (1976), Dockery et al (1981), and Repace (1980) found that the concentration of particulate matter indoors was directly proportional to the quantity of cigarettes smoked. Dockery found that one smoker contributed up to 12 ug/m^3 of RSP whereas more than 2 smokers contributed up to 46 ug/m^3. Repace found that smoking contributed significantly more (up to 600 ug/m^3). Weber (1980) found that smoking contributed up to 100 ug/m^3 to the domestic loading for particulates.

Wade et al, (1975) showed that homes with gas stoves had a NO_2 source independent of outside NO_2. In fact, NO_2 so generated, diffused to other parts of the home, for example, in the kitchen close to the stove the NO_2 concentration was 100 ug/m^3 whereas it was 61 ug/m^3 and 52 ug/m^3 in the living room and bedroom respectively. This is expected since the combustion of natural gas results in the production of oxidized products which include SO_2, NO_2 and CO; the NO_2 coming from the oxidation of the nitrogen in the atmosphere. Goldstein et al (1979) found that NO_2 was higher in kitchens with gas stoves (5-317 ug/m^3) compared to kitchens with electric stoves (6-188 ug/m^3); and NO_2 was also higher in bedrooms of homes with gas stoves i.e. 4-169 ug/m^3 versus 3-37 ug/m^3 than in homes with electric stoves.

Aerosol spray cans could be another source of irritant particulates and gases (Berres et al, 1976). The particulate fraction may consist of waxes, resins, surfactants, corrosion inhibitors and perfumes; the gaseous fraction may consist of aliphatic and chlorinated hydrocarbon propellants, carbon dioxide, and perfumes (Bridbord et al, 1975).

Other internal sources of pollution could be fireplaces, wood stoves, gas heaters, humidifier systems, ageing of surfaces, dried and scaling epidermis, hair and foods. Sofoluwe (1968) found in Nigerian kitchens, where wood was burned, mean levels of NO_2 and SO_2 at 170 mg/m^3 and 300 mg/m^3 respectively. These are extremely high levels and may represent an error in measurement since he used gas detector tubes which are inaccurate and sometimes non-specific. Dockery et al (1981) indicated that the increased sulphate levels which were associated with smoking indoors may have been due to the use of high sulphur matches.

2.2 Effects of Individual Pollutants

2.2.1 Sulphur Dioxide

(a) Formation

Sulphur dioxide is produced from the combustion of coal, natural gas, petroleum, and to a lesser extent from the smelting of lead, iron, zinc and copper, and in the production of H_2SO_4 (Robinson et al, 1970). Its formation results from the oxidation of sulphur and sulphides (inorganic and organic) at elevated temperatures. Other sulphur products are formed e.g. H_2SO_4, MSO_4 (M = metallic cation) and SO_3. Sulphur dioxide takes part in photochemical reactions but its activity is dependent on the intensity of ultra violet radiation and on the presence of other reactants such as NO_2, olefins, moisture and ozone, (Cox et al 1970; Buffalini et al, 1971; Wilson et al, 1972; Sander et al, 1976; Harker, 1977).

(b) Effect of SO_2 on Humans

Sulphur dioxide exposure results in bronchoconstriction which is manifested by an increase in airways resistance (Frank et al, 1962). Speizer and Frank (1966) compared the resistance with nose and mouth breathing for ten minute exposure periods at 43 mg/m^3 and 80 mg/m^3. Airways resistance was greater when SO_2 was inhaled by mouth than by nasal inhalation. No explanation was advanced for these observations; these authors noticed, moreover, that the resistance had increased in

the nose breathing subjects after a fifteen-minute interval. One explanation for this phenomenon may be the high solubility of SO_2 in aqueous media. More of the SO_2 is dissolved in the upper respiratory tract and nasal passages, resulting first in a very small pulmonary response but, as the gas enters the tissues and the blood stream, it is re-circulated to the pulmonary airways, resulting in the delayed reaction as observed by Frank and Speizer. Mouth inhalation results in lower absorption and therefore the more immediate response and faster recovery.

Lawther et al, (1975) used deep breathing to overcome absorption in the upper respiratory tract and found increases in airways resistance at concentrations between 3-9 mg/m^3. Kreisman et al (1976) found a decrease in lung function using maximal expiratory flow rates at 40% of vital capacity (MEF40%) at concentrations between 3-15 mg/m^3 during light exercise. The response at the lower concentration (3 mg/m^3) was variable and symptomatology at 9 mg/m^3 included cough, dryness, irritation and burning of the throat.

Other researchers have found the same types of responses in challenge studies of short duration at concentrations greater than 3 mg/m^3 (Frank et al, 1964; Snell et al, 1969; Andersen et al, 1974). Andersen et al (1974) found also a decrease in mucus flow rate in the nasal passages; these researchers observed that mucostasis may account for the manifestation of common cold symptoms by four of the fifteen subjects since this response (mucostasis) results in a breakdown of the normal defence mechanism against microbiologic and toxic agents. This

may be only one explanation for this occurrence since Zarkower (1972) found that there was a decrease in antibody production on exposure to SO_2 alone or to SO_2 and carbon combined. This phenomenon may also result in increased respiratory infection, and it is likely that both mucostasis and decreased antibody production occur on exposure to SO_2.

Sheppard et al (1980) showed increased airways resistance at 2.5 mg/m^3 in asthmatics by mouth breathing, and in a repeat study found responses at levels as low as 1.5 mg/m^3 (Sheppard et al 1981). Jaeger et al (1979) showed, with mouth breathing, a small decrease in maximal flow rate at 1.5 mg/m^3. Koenig et al (1980) used asthmatic subjects and found a decrease in maximum expiratory flow rate with a mixture of SO_2 and sodium chloride. A repeat study (Koenig et al, 1981) using subjects with exercise induced bronchospasm, also showed a response with SO_2 and sodium chloride. Other researchers found similar responses at similar concentrations in asthmatic and bronchitic subjects (Reichel, 1972; Weir and Bromberg, 1973; Bell et al, 1977; Bedi et al, 1979; Kagawa and Tsuru, 1979; Islam and Ulmer 1979a, 1979b; and Kleinman et al 1981).

c) Laboratory Animal Studies with SO_2

High concentrations of SO_2 (572 to 1330 mg/m^3) produced lesions closely resembling chronic bronchitis in dogs (Knight et al, 1974), rats, and hamsters (Lamb and Reid, 1968). Studies using low levels of SO_2 (0.4 to 2.8 mg/m^3) over an extended period (78 weeks) showed no changes in lung function or blood chemistry (Alarie et al, 1970, 1972).

Laboratory animals react to SO_2 by constriction of the large and small airways (Amdur, 1969). It has been shown that exposure to SO_2 induced the production of histamine in the lung. Nadel et al (1965) were able to show alleviation of SO_2-induced constriction by the use of an anti-histamine. P'an and Jegier (1970) found that SO_2 inhibited acetylcholinesterase enzyme activity at 14 mg/m^3. Barry et al (1970) found that SO_2 at about 900 mg/m^3 resulted in hypersecretion of mucus in the lower respiratory tract and a marked increase in acid phosphatase activity in alveolar cells. In the Yokoyama and associates (1971) studies with radio-labelled SO_2, there was an increase in alpha-globulin levels. Petering and Shih (1975) suggested that SO_2 exposure may result in an increase of bisulphite and thiosulphate in the plasma and inorganic sulphate in the urine. Knauss et al (1976) found that exposure to extremely high levels of SO_2 (1500 mg/m^3) resulted in an increase of mucus and cellular material in the alveoli, and a decline in the goblet cells in the bronchioles on initial exposure with a significant increase thirty hours after exposure.

2.2.2 Nitrogen Dioxide (NO_2)

(a). Formation

Most of the anthropogenic NO_2 comes from the combustion process whereby atmospheric nitrogen is oxidized by atmospheric oxygen at high temperatures. Bacteriologic action on nitrogenous material in the soil results in the production of NO which is rapidly converted to NO_2. This latter production accounts for ten times the NO_2 that emanates from anthropogenic sources (Robinson and Robins, 1970).

(b) Human Exposures to NO_2

Exposure of chronic bronchitics to 3.0 to 3.7 mg/m^3 of NO_2 for 15 minutes produced a detectable increase in airways resistance (Von Nieding et al, 1973). After exposure to 9.4 mg/m^3, normal subjects exhibited decreased diffusing capacity of the lungs (Von Nieding, 1972). Orehek (1976) evaluated the combined effect of NO_2 and a bronchoconstricting agent and found that bronchial sensitivity increased when NO_2 was used with the agent in contrast to the agent and clean air.

Kerr and associates (1979) found that asthmatics reported more symptoms on exposure to $1mg/m^3$ NO_2. A control group showed no similar responses. The lung function measurements on both groups showed no significant changes following exposure.

(c) Laboratory Animal Studies with NO_2

Much more research has been conducted with NO_2 on animals than on humans. Acute and chronic exposures to NO_2 at 80 mg/m^3 appeared to have widespread effects on O_2 diffusion and enzyme activity (Buckley and Balchum, 1965). Boren (1967) found that inhalation of carbon particles and NO_2 resulted in macrophage immobilization and tissue destruction. When the order of exposure was reversed, the tissue response was less. This suggested that prior carbon exposure caused a destruction of the defense mechanism, thus allowing the NO_2 free access to the alveolar spaces. This has important implications in that

both pollutants are present in the home atmosphere.

The evidence for macrophage destruction by NO_2 explains the findings of increased infection following exposure to bacteria (Ehrlich and Henry, 1968). Chronic exposure of mice to very low levels (1 mg/m^3) resulted in increased infection and mortality. The tissue and cellular responses were further elucidated using electron microscopy (Stephens et al, 1972). Rats exposed to concentrations of NO_2 (34 mg/m^3) showed a loss of cilia, thickening of tissue, epithelial injury adjacent to the terminal bronchioles, destruction of type I cells and repair occurring with cuboidal (type II) cells. At 4mg/m^3 the following responses were evident: loss of cilia, hypertrophy, and hyperplasia in the epithelial cells of the terminal bronchioles. Termination of exposure resulted in a return to normal after twenty one days. In this same series of studies, Evans et al, (1972) stated that the type II cells may be responsible for surfactant production and macrophage production. NO_2 had also been implicated in surfactant reduction (Arner and Rhoades, 1973).

2.2.3 Total Suspended Particulates (TSP)

(a) Formation

Airborne particles consist of re-suspended matter, vegetable fibres, metals and metallic compounds, organic matter, bacteria, pollen, spores, molds, water drops and hydrated compounds.

Suspended particulate matter in the atmosphere falls within the size range of 0.1-100 um. The distribution of particle size usually follows a log-normal distribution. Particles smaller than 0.1 um undergo Brownian motion, i.e. a random motion colliding with other particles or molecules which may result in particle growth. Particles larger than 1 um have significant settling velocities and obey Stoke's law (i.e. settling velocity is proportional to the product of the square of the particle diameter and the particle density and inversely proportional to air viscosity). Particles larger than 5 um are removed from the atmosphere by gravity. Particles smaller than 1 um are formed by condensation reactions in the atmosphere. Combustion of fuels produces particles of a broader size range than the condensation reactions.

McCuster et al (1981) found that the mass median diameter of cigarette smoke was 0.52 to 0.67 um, and for cigars it was 0.40 to 0.46 um. Lefcoe and Inculet (1975) found that particles from smoking were less than 1 um. Berres et al (1978) found that the use of air refreshener propellants produced particles in the size range of 1.0-6.2 um.

(b) Human Responses to TSP

Although airborne materials have a broad size range reaching 100 um. in diameter, most particles reaching the pulmonary air spaces are usually less than 5 um. The Task Group of Lung Dynamics (1966) proposed a model for particle deposition in the lung by dividing the lung into its three main compartments i.e. nasopharyngeal, tracheobronchial and pulmonary. They also made these assumptions: (a) a log-normal distribution of

suspended particles. (b) a constant breathing rate of 15 breaths per minute. (c) aerodynamic properties of the particles. The model showed that there was very little deposition in the pulmonary spaces of particles greater than 10 um in diameter. The three mechanisms of deposition operating in the lung are impaction, gravitational settling and diffusion. Inertial impaction is of major importance in the upper respiratory tract and therefore pertains to the larger particles. Gravitational settling is dependent on particle diameter and density and is of importance in the lower respiratory tract.

Diffusion occurs in the pulmonary spaces where air velocities are lower and distances to the tissue surfaces are less.

Challenge studies using the normal composition of town air or the air in domestic environments have not been attempted. There are obvious difficulties since the chemical and physical composition of airborne particulates will vary from site to site and day to day. Koenig et al (1980 and 1981) showed that SO_2 in combination with sodium chloride caused a decrease in maximal expiratory flow rate at 50% and 75% of vital capacity in asthmatics, but this study cannot be equated to typical conditions in the town or in the home since sodium chloride is not a normal constituent of the atmosphere. Numerous epidemiological studies have shown that airborne particulate matter does cause respiratory diseases, and these will be discussed in Section 2.3. Frank (1980) reviewed the interaction between SO_2 and TSP and inferred that the combined effect on respiratory health is greater than that of the pollutants individually.

(c) Animal Responses to TSP

The inhalation of smoke alone by mice (50 mg/m^3) resulted, after thirty six hours of exposure, in no fatalities and no signs of edema, haemorrhage or emphysema (Pattle, 1957). Amdur (1952, 1963, 1971) showed that exposure to particulate sulphate, sulphuric acid mist, and SO_2 with sodium chloride aerosols resulted in increased airway resistance. Boren et al (1967) found that exposure to carbon particles and NO_2 resulted in pulmonary edema and macrophage destruction.

2.3 Epidemiological Studies on the Effects of Air Pollution in the Domestic Environment

In reviewing epidemiologic studies in which the causal factors are being sought for chronic disease, one has to determine the degree of uniformity between all of the relevant factors. In this section, studies are described which cover many countries and where the researchers used a variety of different measurement techniques.

The British Medical Research Council (MRC) Questionnaire on respiratory diseases was developed in the early 1960's and has been widely used internationally. The Dutch have modified certain of the MRC questions to produce the TNO (Applied Science Research Institute) Questionnaire. The Americans have also modified and expanded the MRC Questionnaire to produce the NHLI Questionnaire (National Heart and Lung Institute). Hence when this common measurement tool is used, it is possible to make comparisons between studies.

Prior to 1970, a wide spectrum of air pollution measurement methods were used. Since then, the Environmental Protection Agency in the USA has developed standardized methods for sampling and analyses. Most researchers have since adopted these methods. However, many of the large classical studies were conducted prior to these standardized methods becoming available.

The other measurement tool is lung function assessment. There has been no widespread adoption of standardized techniques. Researchers have used and are still using the peak flow meter, the wedge spirometer and the bellows spirometer. Electronic spirometers have only been introduced recently. In the main, the forced expiratory flow volume in one second $(FEV)_1$, the forced vital capacity (FVC) and the peak expiratory flow rate (PEFR) were the function measurements widely used. More recently, measures of small airways calibre such as the maximum expiratory flow at 50% of vital capacity ($MF_{50\%}$) and the maximum expiratory flow volume at 25% of vital capacity ($MF_{25\%}$) have come into use. Normal values for all of these measurements have become available and have been widely used internationally (Morris et al 1971; Cherniack and Raber 1972; Schoenberg et al 1978).

2.3.1. Air Pollution and Mortality

Widespread recognition of the effect of air pollution on health occurred during the extremely high air pollution levels following meteorologic conditions which allowed minimal dilution and dispersion. Sixty three excess deaths occurred in Belgium in 1930 (Firket, 1936); 20 in Pensylvania in 1940 (Shrenk, 1948); and 4,000 in England in 1952

(Wilkins, 1952). Buechley et al (1973) associated increased SO_2 with 2% excess deaths in New York from 1962-1966, and Winkelstein et al (1967, 1968) found an association with increased particulate loading and mortality of white men and women over 50 years of age. Biersteker and Evendijk (1976) found that increased mortality in Rotterdam was associated with temperature rather than pollution.

2.3.2 Air Pollution and Exacerbation of Respiratory Deseases

Subjects with existing respiratory diseases may be classified as a high risk group since they may be more sensitive than normal subjects to air pollution exposure. Studies using this group of subjects may be useful in setting exposure standards for outdoor air pollution levels.

Spicer et al (1976) found that in a group of seminary students with chronic obstructive pulmonary disease, the only factor which explained the changes in lung function was air temperature. Air temperature was also given as the primary factor responsible for increased reporting of complaints in a group of factory workers with chronic bronchitis (Gregory, 1970), and a group of bronchitic patients in the City of Westminister, London (Emerson, 1973). Skoog et al (1976) found that patients showed improved lung function on moving from an area of 0°C to one of 21°C.

Numerous researchers used diaries to determine symptom changes as a function of air pollution changes. Lawther et al (1970) found that symptom reporting increased as the levels of air pollution increased to

500 ug/m^3 for SO_2 and 250 ug/m^3 for TSP, the response being greatest in early winter. Burrows et al (1968) found that SO_2 and air temperature explained most of the worsening in respiratory symptoms, and Wever (1977) found an association between respiratory symptoms and nitric oxide however nitric oxide is not a respiratory irritant. Carnow et al (1969) found an increase in the reporting of symptoms in a group of chronic bronchitics when SO_2 levels went above 100 ug/m^3. Zagranski et al (1979) found no symptom changes with air pollution changes on a group of asthmatics in New Haven, Connecticut.

2.3.3 Air Pollution and Acute Respiratory Changes in Normal Subjects

Epidemiological studies using random by selected normal subjects may be useful in determining the kinds of pollutants that may cause diseases of the respiratory tract. It is thought that repeated air pollution episodes and hence repeated irritation may lead to permanent changes in the respiratory tract (Fletcher, 1977; Colley, 1974).

In this series of studies, with normal subjects, air temperature was found to be associated with increased reporting of respiratory symptoms and decreased lung function (Spodnik et al, 1966; Spicer and Kerr, 1970; Ipsen et al, 1969). Durham (1974) found that air pollution and air temperature accounted for the increased reporting of symptoms, and Hammer et al (1974) found an increase in cough in a group of nurses exposed to outdoor levels of 600 ug/m^3 of ozone, but no other respiratory symptoms when levels were below the 600 ug/m^3 level.

Lawther et al (1974 a, 1974b, 1974c) studied lung function changes in a group of four subjects over a five year period. Maximum mid-expiratory flow (MMEF) was inversely associated with air pollution in three subjects, but all the other lung function measures for the group showed inconsistent association. In one subject (Lawther et al, 1973), there was a good correlation between SO_2 and airways resistance, but the three other subjects did not respond in the same way (Lawther et al, 1977).

Air pollution episodes have been associated with increases in respiratory infections. This was found by Cassel et al (1972) in a New York study, by Verma et al (1969) in another New York study, and by Shy et al (1970) in a Tennessee study where the increased infection rate was associated with increased levels of NO_2. Kalpanazov (1970) found increased respiratory infections following an air pollution episode in Bulgaria. Monto and Ross (1978) found that bronchitic subjects had more frequent bouts of respiratory infections, and these researchers rationalized that repeated respiratory infections may be a precursor to chronic respiratory diseases. Other researchers have implied that this association exists. (Coffin et al, 1968; Fletcher et al 1977; and Lebowitz et al, 1978; Yarnel and St. Leger, 1981).

2.3.4 Air Pollution and Chronic Respiratory Diseases

I separated the literature into longitudinal studies and cross-sectional studies. The findings from these studies are summarized in Appendix 1.1 and Appendix 1.2 respectively. This tabular method was chosen because of the need to summarize a large mass of information.

In general, urban residence was associated with increased symptom reporting and decreased lung function (Anderson et al, 1965a; 1965b; 1965c; 1965d; Ferris et al, 1965; 1971; 1973; Mostardi et al, 1974; Petrilli et al, 1966; Aubry et al, 1979; Neri et al, 1975; Holland et al, 1965; Colley et al, 1967). In some of the studies, the urban-rural gradient was not seen (Comstock et al, 1973; Holland et al, 1969). In three California studies (Cohen et al, 1972; Linn et al, 1976; Detels et al, 1979) the researchers attempted to assess whether chronic O_3 exposure resulted in chronic diseases, but no consistent associations were found between ozone levels and chronic diseases.

In most of the above studies there were many methodologic short-comings which included: lack of consideration for confounding factors; improper exposure data; and limited attention to non-respondents. Occupational and domestic exposures were occasionally mentioned, but details were not given on their assessments. In some of the towns chosen, although a gradient of air pollution existed, in many cases the levels were not high enough to cause physiologic changes between residents. In one case, the results seemed to be overstated (Ferris et al, 1964, 1971 and 1973). In the majority of cases, univariate analyses were conducted.

2.3.5 Air Pollution and Respiratory Health of Children

Studies using children are of benefit in determining causal factors in respiratory diseases in that the confounding factors of smoking and occupation are absent, their residential histories tend to be unvaried, participation rates are high, and their respiratory systems may be more

sensitive to external insult. There are some difficulties though:

(a) children may not be able to perform the lung function tests in a reproducible manner,

(b) normal lung function values may not be available for all age groups,

(c) the growth of the lungs and improvement in air pollution controls may make it difficult to detect changes, and

(d) parents are usually asked to complete questionnaires on behalf of their children and this may produce bias in reporting (Schilling et al, 1977)

Many researchers have studied children and these have been summarized in a tabular fashion in Appendix 2.1 (longitudinal studies) and Appendix 2.2 (cross-sectional studies).

These studies have shown that children are in fact a sensitive test group to determine the factors in respiratory disease causation. It has been shown that urban-rural differences are more evident in children, and more specifically, children seemed to respond to levels of air pollution which were lower than those seen in studies where adults were the subjects (Holland et al, 1969a; Holland et al, 1969b; Paccagnella et al, 1969; Stebbings et al, 1976; Lunn et al, 1967; Colley et al, 1974; Lebowitz et al 1974; and Shy et al, 1973).

The response rates were very high (i.e. in excess of 93%) in most cases where total populations were studied (Kerrebijn et al, 1975; Holland et al, 1969a; Holland et al, 1969b; Colley et al, 1970; Colley et al, 1974). In the longitudinal studies, the participation rates decreased with time (Douglas et al, 1966; Colley et al, 1973; Kiernan et al, 1976).

2.3.6 Domestic Factors and Respiratory Diseases

In Section 1.2 of this thesis, nine domestic factors were selected for study. This review of the literature will be organized according to seven of these factors since I was unable to find any references on the effects of pets and hobbies. Some other factors that were not included in this study were also reviewed for completeness.

(a) Heating fuel

Yarnell et al (1979) studied a random selection of residences in a coal mining area in the United Kingdom. The residences were divided into those with central heating and those with coal fireplaces. More asthmatics were found in homes with coal fires, but residents in these homes had better FEV_1 compared to those who lived in homes with central heating. In a statistical study to evaluate the contribution of domestic heating on mortality, Lave and Seskin (1972) found a statistical correlation between mortality and the kind of heating fuel used. This study also showed that when the domestic heating variable was added to the multiple regression analyses, the outdoor air pollution variables tended to lose significance.

(b) Cooking fuel

In studies designed to test specifically the contribution of domestic exposure, Keller (1979a, 1979b) found in an urban population in Ohio, USA, that electric stove users had marginally more respiratory infection than gas stove users. There were no differences in the lung function of residents between homes despite differences in NO_2 levels as measured by the Jacob-Hochkeiser method (100 ug/m^3 for gas users vs 40 ug/m^3 for electric). In a similar study conducted in Britain and reported by the following researchers, (Melia et al, 1977, 1979; Goldstein et al, 1979; and Florey et al, 1979), higher prevalence rates for cough, colds and bronchitis were found in boys and girls who lived in homes with gas stoves. In this study, 38 schools were randomly selected in the poorer areas of England. Of the total 9,128 students, 5,728 participated. Questions were not asked on parental smoking or crowding or other domestic factors. The original study started in 1972, and in 1977, a follow-up study indicated similar association between gas stoves and respiratory health, but the absolute differences were smaller. From the main study group, 808 students were selected to determine the correlation between lung function and NO_2 levels in the home; but no statistical associations were found. The NO_2 was measured by a passive diffusion method which is known to be insensitive to low levels of NO_2.

Hasselblad et al (1981) studied 5 to 13 year old children using questionnaires and lung function tests. The use of gas stoves was found to have a small effect on the FEV_1 of girls. Comstock et al (1981)

found in an adult population in Washington that in homes with gas stoves, the male population had more cough, wheeze and dyspnea than males who lived in homes with electric stoves; there were no such trends in females. Jones et al (1981) found a weak association between gas cooking and FEV_1 (RR=1.29) in a group 20-39 year old, non-smoking, Michigan females. Dockery et al (1982) found that the effect of gas stoves on the FEV_1 of children was small, and maternal smoking together with gas stove use resulted in a decrement of about 20 ml for FEV_1. Dodge (1982) reported that children living in homes with gas stoves had more cough, but not lower lung function when compared with children living in homes with electric stoves.

Two studies were reported from developing countries where wood stoves were used. Sofoluwe (1968) found children with bronchiolitis and bronchopneumonia in homes with extremely high levels of NO_2 and SO_2 (the method of assessing exposure was questionable). Anderson (1978) found no asthma or wheezing in highland residents in New Guinea who used wood stoves.

(c) Humidifiers

Humidifiers can be sources of microbial aerosols that may cause increased infections which may lead to permanent respiratory diseases. Fink et al (1976) found the cause of an outbreak of acute respiratory distress to be due to airborne fungi from a humidifier reservoir. Hosein (1976) found similar symptoms in two workers who used a humidification system as part of an industrial process. Korsgaard

(1982) found that house mite concentrations were higher in homes with high humidity, and the homes of patients with house dust allergies had higher humidity. Holma (1980) reported that in older Danish homes, dampness accounted for some of the excess symptoms in adults.

(d) Air Conditioning

In the multiple regression analysis by Lave and Seskin (1972), a negative correlation was found between air conditioning usage and mortality, but when climate was added as a variable in the analysis, air conditioning lost its significance. This indicated that air conditioning may be acting as a surrogate for the climate variables.

(e) Crowding

Reid et al (1958) investigated the effect of domestic crowding on respiratory health. They found that crowding was associated with increased coryza (nasal production of phlegm). Holland et al (1960), Colley et al (1974) and Leeder et al (1977) have all stated that domestic crowding may be a possible factor in respiratory disease causation. Davis and Bulpitt (1980) found in a small group of general practice patients that families of four or more persons had more wheeze and atopy than smaller sized families. This may be a result of a common domestic exposure factor or it may be due to congenital factors.

(f) Passive smoking

Colley et al (1974) indicated that parental smoking had a small effect on the respiratory symptoms of children, and an editorial in the Lancet (1974) explained that such findings may not be due to direct exposure to cigarette smoke but to indirect means such as cross-infections. The suggestion is that parental smoking may result in parental respiratory disease which may spread to children (Monto et al, 1975). Leeder et al (1976) also stated that the familial associations in chronic disease may be due to parental cigarette smoking indoors. Tager et al (1976) found similar familial associations but they indicated that parental smoking was of minor importance. In another study by these authors (Tager et al, 1979, 1982), a dose response relationship was found between lung function score and the number of smokers in the home. In a follow-up of this study (Weiss and Tager, 1980), parental smoking resulted in significantly more wheeze in children, but the findings for cough and phlegm production were less consistent; wheeze in children was found to be independent of parental wheeze. Hasselblad et al (1981) found that maternal smoking had a small effect on the FEV_1, of boys, but not on the FEV_1 of girls. Yarnel et al (1981) found that continuous infection resulted in decreased lung function in children, and the effect of passive smoking on lung function was absent.

Lebowitz et al (1970) studied 3,484 white subjects in 1,655 households and found that children under 15 years old, who lived in homes with smokers, had more symptoms than to those who lived in homes of non-smokers. In older children, similar trends were not seen. Also in

this study, the researchers showed that children, who lived in homes with parents with respiratory symptoms, had a higher symptom prevalence rate compared with those who lived in homes with symptom-free parents; this association was independent of smoking.

Harlap (1974) reviewed the admission to paediatric wards in Jerusalem and showed that children of smoking mothers made more visits compared with those of non-smoking mothers. These children also had higher rates for bronchitis, pneumonia and lower respiratory infection; there appeared to be a dose response with the number of cigarettes smoked by the mother. Ferguson et al (1980) studied a cohort of infants at birth, at 4 months and at 12 months, and found that upper and lower respiratory tract infections were associated with maternal smoking, the effects being independent of perinatal history and home background. Shenker et al (1981) studied 4,071 children using spirometry and a questionnaire and found that parental smoking was associated with increased reporting of chest illnesses; the associations being strongest for maternal smoking and for girls, and for children less than 2 years old. Cameron et al (1969) found in a Detroit population drawn from the telephone directory that children 5 to 16 years old, who lived with smokers, had more acute respiratory infection that those who lived with non-smokers; similar effects were not seen with children less than 5 years old. In a follow up to this study (Cameron and Robertson, 1973), which was expanded to include residents of Long Beach and Pasadena, it was found that the children in homes with smokers had more acute respiratory infection than those in homes with non-smokers. Binder et al (1976) found that although the homes of smokers had more TSP, this was not associated with excess reporting of symptoms by children.

Comstock et al (1981) and Meyer et al (1981) reported on their study of a group of residents of Washington, D.C. They found that non-smoking adults who lived in homes with smokers had a higher relative risk for cough, phlegm and wheeze (females only), and dyspnea than non-smokers in non-smokers' homes, but none of these findings was statistically significant. In a longitudinal study by White and Froeb (1980), it was shown that non-smoking adults who lived in homes with non-smokers had better forced expiratory flow (FEF) than non-smokers living in non-smoking homes but who were exposed to smoke at work. In this study some bias may be expected since all subjects were participants of a fitness group who volunteered to take part in this respiratory disease study. Dockery et al (1982) found in the six-city Harvard study that passive smoking had an extremely small effect on the lung function of children. Dodge (1982) reported that parental smoking was associated with cough and wheeze in children. Kauffmann (1980) and Kauffmann et al (1983) found that in older women, passive smoking was associated with lower forced expiratory flow.

Passive smoking was shown to be associated with cancer in two recent studies. Hirayama (1981) showed in a prospective study of 91,540 non-smoking housewives, that those whose husbands were smokers had an increased risk of developing lung cancer compared to those whose husbands were non-smokers. It was also found that in agricultural families the risk was higher; and the mortality from asthma and bronchitis was associated with spousal smoking, but the differences between the smoking groups were not statistically significant. In the second cancer study Trichopoulos (1981) looked at non-smoking females

with lung cancer. It was found that cases had more husbands who were smokers e.g. the relative risk was 2.4 for those whose husbands smoked less than 20 cigarettes per day and 3.4 for those whose husbands smoked more than 21 cigarettes per day. The presence of polycyclic aromatic hydrocarbons in cigarette tar may account for some of these excess cancers, but some researchers have implicated radioactive particles which may be absorbed into the tobacco leaves from the soil (Robertson and Rogers, 1980; Winters and DiFranza, 1982).

(g) Other Domestic Factors

Many researchers tried to determine whether the use of spray aerosols within the home could contribute to respiratory disease. Zuskin et al (1974) found a small but significant reduction in maximum expiratory flow rate in test subjects following inhalation of hair spray. Friedman et al (1977) on the other hand, found no changes in lung function in a challenge study, but found that some subjects had lowered mucus velocity and chest tightness. Lebowitz et al (1970) found in an epidemiological study that aerosol usage was associated with respiratory symptoms, and females were found to have more chronic obstructive symptoms probably as a result of exposure to spray aerosols.

Spivey et al (1979) studied black children in Baltimore, Maryland, using questionnaire and lung function tests. These researchers observed that residents of public housing had more symptoms and lower FEV_1, than residents of private housing.

2.4 Summary

The domestic environment was shown to be a possible source of a variety of airborne contaminants which, when inhaled over extended periods of time, may lead to respiratory symptoms and depressed lung function. Challenge studies on animals and human volunteer subjects showed that exposure to individual pollutants can result in a variety of respiratory tract responses, but in many cases, the positive responses were associated with pollutant levels well in excess of those normally encountered in the ambient environment.

Epidemiological studies have shown that episodes of high air pollution levels resulted in aggravation of existing diseases, but very often, air temperature was found to explain more of the physiologic responses. In healthy subjects, air temperature was also shown to be important in explaining some of the respiratory responses. Chronic exposure studies using urban-rural comparisons showed that when the urban-rural gradient was large enough, urban residents reported more symptoms and showed depressed lung function. Some studies on the other hand showed no differences in respiratory health despite chronic exposures to high air pollution levels. In many of the epidemiological studies using outdoor air pollution data, the researchers indicated that assessing the domestic exposure may be more sensitive since the outdoor exposure data were not sufficient enough to explain the diseases that were observed (Spengler et al, 1981; Holland et al, 1969[b]).

The use of gas stoves was associated with high levels of NO_2 which in turn was associated, in some cases, with excess respiratory illnesses or diseases. Passive smoking was associated in some cases with increased infection, more symptom reporting, depressed lung function, and high cancer prevalence rates. Children seemed to be hypersensitive to air pollution exposure.

Air pollution exposure was implicated in causing increased respiratory infection. Some researchers feel that repeated infections may lead to disease and this is especially important in the domestic environment because of exposure to common air pollutants and the potential for cross-infection (Colley, 1974; Fletcher et al, 1977; Monto et al, 1977).

CHAPTER 3

METHODS

3.1 Selection of Study Population

The study reported here was part of a larger study to determine the effects of a number of risk factors on respiratory diseases using a well tested, standardized questionnaire and an automated electronic lung function testing system. The risk factors studied were: family history, urban-rural residence, smoking, occupation, sensitivity to allergens, domestic exposure, outdoor air pollution exposure, and climate. As a consequence the populations were chosen from three towns with these characteristics:

(a) one had an urban and another had a rural character

(b) the population size in each was in the vicinity of 4,000

(c) one of the population groups lived in a climate zone different from the comparison group (Figure 3.1)

(d) the occupational histories of the populations were different enough for meaningful comparisons

Two Connecticut towns and one South Carolina town were chosen that met the above criteria. One of the Connecticut towns, Ansonia, situated in the Naugatuck Valley was reported by the State Environment Authority to have the highest air pollution in the State over a number of years (State of Connecticut, 1971, 1973). The annual average total suspended particulate

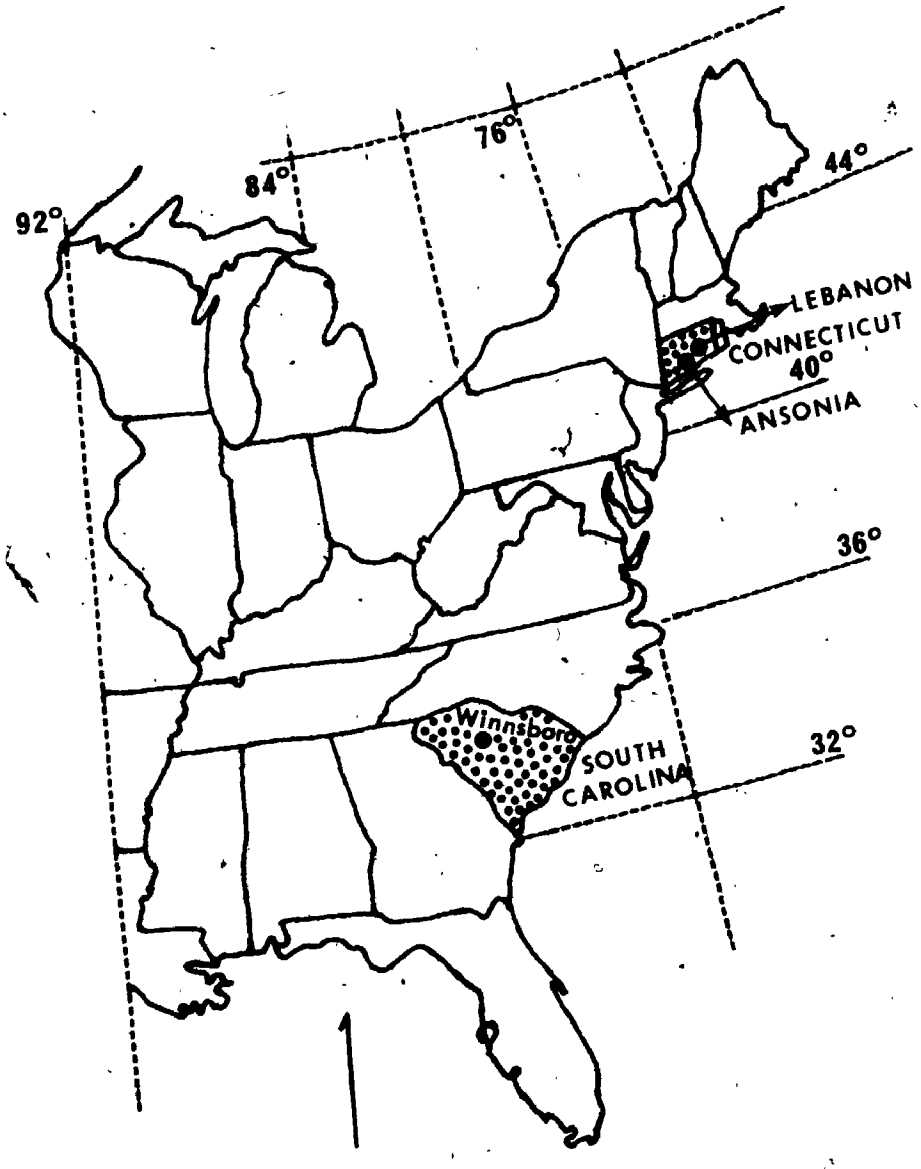


Figure 3.1

Map of Eastern United States Showing the Three Towns Surveyed

Table 3.1 - Characteristics of the Three Towns

	Lebanon, Conn. (rural)	Ansonia, Conn. (urban)	Winnsboro, S.C. (semi-rural)
No. inhabitants	3,800	21,200	4,400
Surface area (km^2)	130	18	21
No. dwellings	1,603	4,469	1,150
No. commercial buildings	4	301	195
No. registered motor vehicles	3,382	11,706	1,700
No. of factory buildings	0	19	9
No. dwellings/km^2	12	248	55
No. inhabitants/km^2	29	1,178	210
No. vehicles/km^2	26	650	81
No. commercial buildings and factories/km^2	0.03	17.8	9.7

Data from U.S. Census (1970), U.S. Geological Survey Maps, and Municipal Registries in each Town.

levels for the periods 1968-1972, exceeded the Federal air quality standard for that pollutant. For comparison purposes, this town was considered to be urban and of a high air pollution burden. The other town selected in Connecticut was Lebanon which was in a rural farming belt and air pollution data from a neighbouring town (Windham) indicated that the air pollution burden was among the lowest in the State. For comparison purposes, this town was considered to be rural and of low air pollution burden. The South Carolina town chosen was Winnsboro and the air pollution data available from a neighbouring town (Newberry) showed that the mean SO_2, NO_2, O_3 and TSP levels were lower than Ansonia, and in some cases, similar to Lebanon. (State of South Carolina, 1973).

Ansonia, Connecticut:

The air pollution burden in this town could be due to two factors (a) large quantities generated within the boundaries of the township by industries and (b) fugitive contaminants that may enter the town from other industrial towns in the valley to the north, northeast and southwest. The total population of the six wards was about 21,000 (1970 U.S. Census), but only Ward 4 was chosen for the study because it met the population size criterion mentioned earlier (Table 3.1). This town had a small black population (24%). The characteristics of Ward 4 were described in the paper by Hosein (1977).

Lebanon, Connecticut:

There was no industrial activity in this town and the main occupation was farming and poultry raising. It was spread over a wider area and was in a low air pollution belt (Table 3.1). The town population consisted mainly of Caucasians.

Winnsboro, South Carolina:

This town had a few small industrial plants and also consisted of some farms (Table 3.1). Some residents were engaged in the cotton textile industry. It had a large black (60%) population. The study of this town allowed an examination of the contribution of geographical and climatological variables to air pollution and lung diseases.

In each town the officials were informed of the study and their participation was sought. Other State and local authorities were advised of the study, and all the local physicians were contacted. Social and cultural groups and clubs were also informed and their help was sought in the census taking and in the canvassing for participants.

3.2 Design of the Population Study

3.2.1 Canvassing

An index card was prepared for each subject in the three study areas from available records including electoral registers, school lists and

telephone directories. The next step was a door-to-door survey in which the card data were checked and subjects were informed of the proposed study. During the conduct of the field study, the canvassing continued by the use of radio, television, the local newspapers, and pamphlets (Appendix 3.1 & 3.2).

3.2.2 Laboratories

Two mobile laboratories were equipped for the conduct of the studies. One served as an office for registration of subjects, recording of their height, and weight and the allotment of subject numbers. It also served as a waiting room. After completion of these preliminaries, subjects entered the second laboratory which housed the lung function monitoring equipment. Included here were two computer terminals connected to a PDP-8E Computer (Digital Equipment Corporation). Each terminal was also attached to a Fleisch 4 Pneumotachograph transducer amplifier system connected to the computer. A hard copy unit (Tektronix Model 4610) was also connected to the terminal. This laboratory housed a body plethysmograph and calibration equipment.

The laboratories were always located on school premises since this afforded not only the security and convenience of electrical power, but it allowed ready access to teachers and all school children over the age of seven years.

3.2.3 Testing

A computer prompted questionnaire was administered to each subject at the terminal and after its completion, the lung function test was performed. Each subject was instructed to expire maximally five times through the pneumotachograph (Figure 3.2 from Bouhuys, 1974) during which flow rates were sampled. Expired volume was obtained by digital integration of flow, and the maximum expiratory flow volume (MEFV) curve was displayed on the terminal's cathode ray tube (CRT). The computer automatically averaged the data for the two blows with the highest forced expiratory volume (FEV_1). This curve was displayed together with a summary of the lung function parameters and questionnaire responses. The lung function variables computed were forced expiratory volume in one second (FEV_1), forced vital capacity (FVC), peak expiratory flow rate (PEFR), maximal expiratory flow at 25% of vital capacity ($MF_{25\%}$) and maximal expiratory flow at 50% of vital capacity ($MF_{50\%}$). A hard copy was made of the information on the display for any immediate use and then filed in the subject's personal file (Appendix 3.3). The data in the computer were stored on punched paper tape and subsequently transferred to magnetic tape for editing and analysis. (See Figure 3.3 for flow diagram).

For the data analyses in the text, the residual lung function measurements were used. Residual (R) = Observed (O) - Predicted (P). The predicted values were derived from the populations studied and included "healthy" participants who had no usual cough or phlegm, no dyspnea, no wheeze, and no history of asthma. The participants used for

developing the predicted equations consisted of non smokers. Prediction equations were developed separately for males and females, blacks and whites, children and adults. The age cut-off for children was 14 for girls, and 17 for boys since at these ages mean height no longer correlated with age. The equations were based on height, age and weight using the multiple regression equations which explained the highest proportion of variation of the dependent variable. Details of the model used were presented in a paper by Schoenberg et al (1978). The population consisted of 3046 participants.

3.2.4 Questionnaire

The British Medical Research Council (MRC) questionnaire on respiratory disease was modified using recommendations of the National Heart and Lung Institute, U.S. Department of Health, Education and Welfare. Nine questions were included in the questionnaire to determine the importance of the domestic environment (Bouhuys, 1974; Appendix 3.4)

Because children of ages less than fifteen years may not remember their medical history, a shorter questionnaire was prepared to be sent to be completed by parents (Appendix 3.5). This included items on past history, symptoms, domestic exposures and residential history. The smoking history was not included in that questionnaire, but instead all children above age 11 were asked the smoking questions from the NHLI questionnaire when they came to the laboratory.

Figure 3.2

Lung Function Test Station Showing

Pneumotachograph, Hardcopy Output,

and Computer Terminal

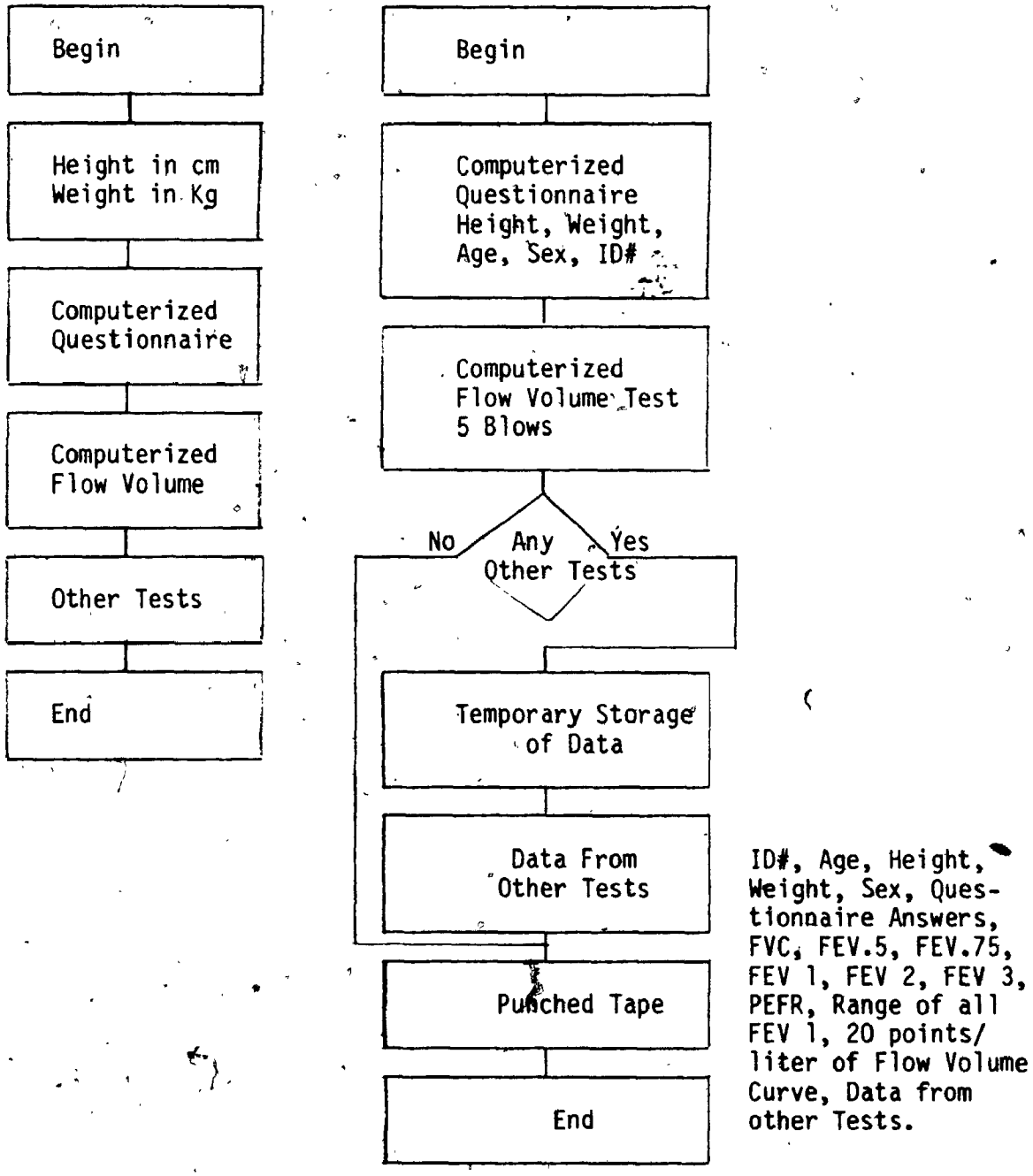


FIGURE 3.3

SUBJECT AND DATA FLOW IN MOBILE LABORATORY

3.2.5 Day-to-Day Management

Registration clerks and interviewers were recruited through the local Lung Associations and hospitals. Selection of the staff was based on a training program given to all applicants and only candidates who were able to perform the task satisfactorily were selected.

The supportive features of the computer freed interviewers from technical involvement and assured that all questions were asked. Feedback from the computer allowed for easy quality control of the lung function tests since the interviewers could have determined from the shapes of the MEFV curves whether there was adequate effort on the part of the subject. Supervision at the laboratory was always undertaken by a senior staff member whose responsibility included equipment calibration, checking of the interview and lung function techniques and answering any medically oriented questions.

Students from the schools were scheduled to perform the test during their free periods. Adults were contacted by telephone from the laboratory and appointments were made for the tests. The laboratory hours were from 9:00 a.m. to 9:00 p.m. on weekdays, and 9:00 a.m. to 5:00 p.m. on Saturdays.

3.2.6 Flow Calibration

The maximum expiratory flow volume equipment was calibrated every two hours. Initially, a 2-liter syringe was used to check the volume output

on the MEFV equipment and the CRT display. Later on, a dynamic calibrator was developed using a cam and piston system to mimic, as closely as possible, a typical MEFV curve.

3.2.7 Selection of Sub-group of Subjects for Domestic Air Pollution Assessment

Housewives were selected for this part of the study because they spend a large proportion of their time at home. They were between the ages of 25 and 44 and included smokers and non-smokers. The symptomatic group answered positively to the questions on "usual cough" and "usual phlegm". These subjects were matched with control subjects of similar age (±5 years), who had similar smoking habits to the subjects with symptoms, but answered negatively to the questions of "usual cough", "usual phlegm" and "ever wheezed". The hard copies (as mentioned in section 3.2) for these subjects were extracted from the files together with the hard copies of all the other subjects in the same house (i.e. children and husbands) (Appendix 3.3).

Because of the restricted definitions used for the selection of study subjects, sufficient subjects were found only in Lebanon and Winnsboro. Only 2 subjects were found in Ansonia and therefore they were not included in the study.

Subjects were called on the telephone, advised that this study was a follow-up of the larger study in which they had participated earlier,

and a mutually agreeable time was arranged for the domestic sampling. The sampler was placed in an area most used by the subject and at a height close to the breathing zone (about 4-5 feet). A short list of instructions was handed to each participant (Appendix 3.6). Subjects were advised to perform all their chores normally and to report any abnormal situations that might influence their exposure over that 24-hour period. A short questionnaire was administered to verify their respiratory symptoms, their smoking habits, the type of heating or cooling system used, the cooking fuel used, hobbies, housekeeping, the presence of smokers, and the health condition of other subjects in the same house (Appendix 3.7). There was agreement between the findings in the short questionnaire and the NHLI questionnaire.

3.2.8 Analysis of the Non-respondents

As part of the overall study each town was divided into up to 10 areas so that comparisons can be made between areas. In order to determine whether non-respondents would have biased the results in any way, non-respondents from one area in each town was contacted in a door-to-door survey to compare symptom prevalences and smoking history of those seen in the laboratory with those not seen. This limited analysis allowed a comparison between non-respondents and respondents in one area, and if there were no differences between respondents in all areas, then generalization could be made about the effect of non-respondents on the findings for each town (Mitchell et al, 1976).

3.2.9 Interviewer Variation

Each interviewer was allocated a personal code which was entered for each subject interviewed. At the end of the survey of the three towns, 14 key questions on symptom reporting and smoking were examined to determine the variation between interviewers.

3.3 Design of the Domestic Air Pollution Sampler

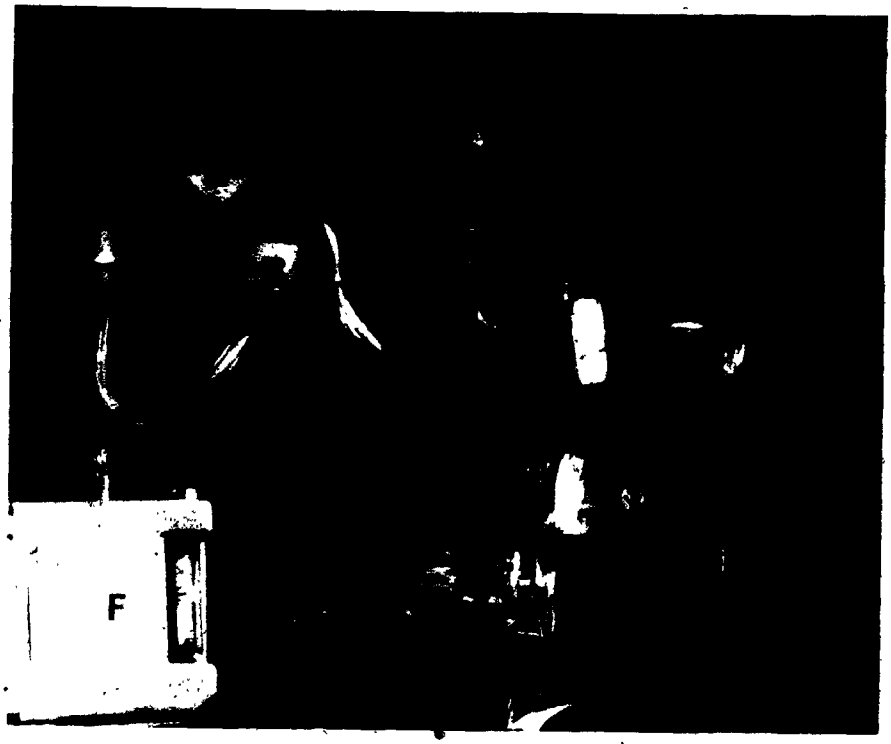
Ambient air quality can be assessed with a variety of stationary sampling instruments. These characterize the air around the instruments but difficulties arise when the results of such measurements are used for an estimate of pollutant loads on people who move around, both outdoors and indoors. To obtain air quality data that might more adequately reflect an individual's changing environment, an easily portable, lightweight, and low-noise air-sampling instrument was designed that allowed individuals to monitor air pollution exposure in their homes for up to 24-hours at one time.

3.3.1 Description of Equipment

The sampler is housed in a 50 x 38 x 15 cm. (20 x 15 x 6") suitcase. In operation, the sampler weighs 7 kg. (16 lbs.). A manifold connected to a blower serves as the main sampling source (Figure 3.4). Probes from the manifold lead to the respective collection media and pumps. A 10-mm nylon cyclone assembly samples respirable-size particulate matter.

Figure 3.4

Personal Environment Sampler for SO_2, NO_2 and RSP



A - Main Manifold

B - RSP Filter (Glass Fiber) with Nylon Cyclone

C - Low Flow Pumps for SO_2 and NO_2

D - Tubes with Absorbing Solutions

E - Main Blower Pump

F - Pump for RSP Sampling

G - Dampener for RSP Pump

H - Flow Meters

The entire housing (suitcase) was lined with closed-cell foam rubber to absorb noise. To aid in sampling for 24-hour periods, a circuit was developed with automatic transfer from DC to AC (line-powered) supply. When the pumps were in the line-powered mode, a trickle-charge was applied to the nickel-cadmium battery for further D.C. operation. Nissei pumps powered by D-size (1.25V), non-rechargeable, Duracell(R) batteries were used to sample for NO_2 and SO_2.

The energy in these batteries decayed linearly over time but remained high for more than 24 hours. For particulate sampling, the manifold diameter and the probe shape, diameter, angle, and position, followed recommended standards, (Lundgren et al. 1967; Sehmel, 1970; U.S. Public Health Service, 1972). To achieve isokinetic sampling, the manifold and probe velocities were matched by increasing the diameters and reducing the flow rates without sacrificing the recommended probe flow rate of 1.7 litres per minute and probe internal diameter of 1.0 cm (Sehmel, 1970).

3.3.2 Sampling Schedule

The homes of the study group were sampled once in the summer and once in the winter. Outdoor pollution sampling was conducted during the same 24-hour period. The samples were analysed according to the methods shown in Appendix 3.8.

3.3.3 Outdoor Air pollution Data

Samplers were strategically placed in each town to define outdoor air pollution levels so that comparisons could be made between towns and between the domestic environment and the outdoor environment. The details of the methods used are shown in Appendix 3.9 and in the paper by Hosein et al (1976).

3.4 Data Analyses

The differences in the symptom prevalence rates between the exposure groups were evaluated using chi-square tests (Armitage, 1974). The relative risk (RR) comparing the rate of disease in those exposed to those not exposed was also determined. The residual lung function between the exposure groups were tested using analysis of variance (Armitage, 1974). The analyses on lung function were conducted on the residuals and on the natural logarithm of the residuals to determine whether the transformation would be more discriminating for the various exposure groups; since no differences were found between the non-transformed and transformed analyses, the results in this text consist only of the non-transformed residuals.

In order to assess whether the exposure factors acted separately to explain the variation in lung function and symptom reporting rates, multiple regression analyses were conducted on the various age, sex and race groups. The stepwise model was used starting first with each of the variables singly, then using the variables which predicted the

highest variation in lung function to develop the two variable models. The pairs of variables which explained the highest proportion of the variation in the two variable models were then used to develop the three variable models. The step-up procedure continued until all models with all nine exposure variables were included. Because five variables were consistently associated with the variation in lung function, I chose to report the findings on the best five-variable model, i.e. the model which gave the best R^2 and the most individual variables which were separately (significantly) associated with lung function.

To determine whether the exposure factors acted in combination with each other, multifactorial analyses of variance were conducted on all of the exposure variables except pets since this variable was shown to have no effect on lung function. This analysis allowed me to determine whether there was interaction between the exposure variables i.e. whether the effects were synergistic rather than additive (Snedecor and Cochran, 1971). All of the data were made into SAS data sets and analysed using the General Linear Model (Statistical Analysis Systems, 1979).

The differences between the mean domestic exposure concentrations of cases and controls were assessed using the Student t-test. One way analysis of variance was used to determine the daily variation between pollutant levels in a special six-day test using two direct reading instruments. Simple and multiple regression analyses were conducted between the pollutant concentrations and lung function measurements to determine the existence of any correlations. (Armitage, 1974; Snedecor and Cochran, 1971).

CHAPTER 4.

RESULTS I

Influence of Domestic Factors on Symptoms and Lung Function.

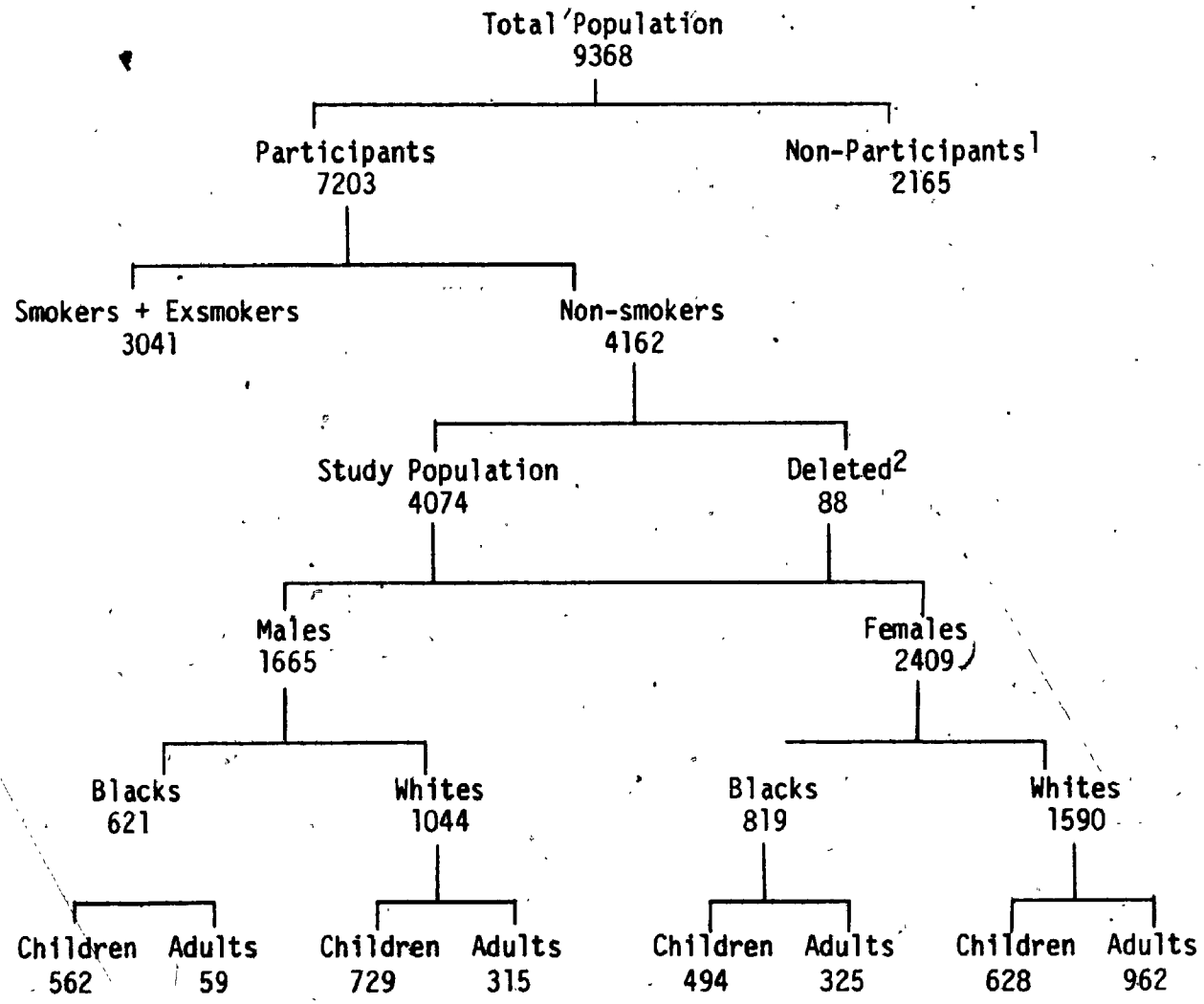
4.1 Study Variables

Only non-smokers were considered in this analysis since the effects of current smoking and previous smoking (Table 4.1) would have had a much greater impact on symptoms and lung function than would the domestic factors. The non-smokers from the three towns were combined for study since it was found that the chronic bronchitis prevalence rates (Table 4.2) and lung function of residents were not significantly different between the towns (Bouhuys, et al, 1978). It was also found that the magnitude of the outdoor pollution differences between towns was small although some of the the differences between the means were statistically significant. (Table 4.3).

The population was stratified according to age, i.e. children and adults, sex and race since these factors have been found to be important in explaining respiratory health (Tables 4.1 and 4.2). The age cut-off used for children and adults was 18 for males and 15 for females. A demographic summary of the study population is shown in Figure 4.1.

DEMOGRAPHIC SUMMARY OF STUDY POPULATION

FIGURE 4.1



1. Interview with a sample of non-participants indicated that overall there were no consistent differences compared to participants for symptoms, smoking habits, working histories, age and sex.

2. These data were deleted because of inadequate exposure data.

TABLE 4.1

THE INFLUENCE OF SMOKING ON CHRONIC BRONCHITIS+
PREVALENCE FOR THE THREE TOWNS (WHITES ONLY)

	Lebanon		Ansonia		Winnsboro	
	N	%	N	%	N	%
SMOKERS						
Male	332	11.7	160	10.6	221	11.3
Female	317	4.1	147	2.7	220	7.1
EX-SMOKERS						
Male	241	4.6	106	6.6	115	4.8
Female	170	1.2	71	5.6	83	0
NON-SMOKERS						
Male	569	1.0	201	2.0	443	1.8
Female	469	1.7	335	1.5	653	1.8

+ Chronic bronchitis is defined as usual cough and usual phlegm for three months per year during two or more years.

TABLE 4.2

PERCENT PREVALENCE OF CHRONIC BRONCHITIS IN NON-SMOKING WHITES AND BLACKS BY AGE AND RESIDENCE

		MALES			FEMALES		
	AGE	LEB	ANS	WINNS	LEB	ANS	WINNS
White							
	7-24	0	0	1.0	0	0	0
	25-44	0	9.1	4.9	3.0	2.3	2.3
	45-64	6.6	3.8	2.6	0.9	4.2	3.8
	65-99	12.5	5.3	10.0	5.4	0	5.8
	Total	1.0	2.0	1.8	1.7	1.5	1.8
Black							
	7-24	-*	1.2	0	-	0.9	0.6
	25-44	-	7.1	0	-	9.2	0
	45-64	-	18.2	8.0	-	0	1.6
	65-99	-	0	9.0	-	0	0
	Total	-	3.9	0.8	-	3.1	0.6

* The number of Blacks in Lebanon was extremely small and hence this rate was not computed.

Chi-square test of prevalence rates between towns showed no significant differences.

TABLE 4.3

POLLUTION DIFFERENCES BETWEEN TOWNS (OUTDOORS)
(geometric mean ± geometric standard deviation)

Towns++	Concentration (ug/m^3) +					
	SO_2	NO_2	TSP	O_3	SO_4	NO_3
Ansonia (A)	7.6±4.2	80.1±1.6	56.4±1.6	85.8±1.5	7.1±2.1	2.4±1.7
Lebanon (L)	5.6±4.6	45.7±1.8	32.8±1.8	90.6±1.3	4.5±2.1	1.7±1.3
Winnsboro (W)	1.5±5.1	31.5±1.6	45.4±1.4	70.7±1.4	5.8±1.7	1.3±1.5
T-tests						
A vs L	*	***	***	***	***	NS
A vs W	***	***	***	***	NS	NS
L vs W	***	***	***	***	NS	NS

* = $P < .05$; *** = $P < .001$; NS = not significant.

++ See Appendix 3.9 for details on outdoor monitoring. Ansonia had 5 sampling sites; Lebanon had 5 sampling sites; and Winnsboro had 3 sites.

+ The mean values reported were mean for each town averaged over the number of sites per town. Samples were collected every sixth day in Ansonia and Lebanon for two years. In Winnsboro, samples were collected on consecutive days for up to 14 days at the peak of each season.

The following variables were considered in this chapter: usual cough (question 16), usual phlegm (question 22), ever wheeze (question 34) and shortness of breath - grade 1 (question 58). The lung function parameters were - r $MF_{25\%}$ (residual maximum flow at 25% of vital capacity measured in liters per second), r $MF_{50\%}$ (residual maximum flow at 50% of vital capacity measured in liters per second), and r FEV_1 (residual forced expiratory volume at one second measured in liters); the domestic factors included pets, hobbies which may cause exposure to gases, vapours and dusts, domestic cooking fuel, fireplace, humidifiers, air conditioning, domestic heating fuel, domestic crowding, and the number of smokers in the home.

4.2 Summary of Univariate Analyses on Domestic Factors, Symptoms and Lung Function in Children and Adults

Nine domestic factors that might influence the growth and decay of the lung were studied individually in a group of non-smoking children and adults. A summary of the univariate analyses on each domestic factor is given in this section. The detailed description of the results and all the univariate tables are given in Appendix 4.1. Some of the domestic factors consistently showed effects on symptoms and lung function, but there were some inconsistencies between the age, sex and race groups, and even between symptom reporting and the lung function measurements. The inconsistencies may be due to potential confounding and interaction between the variables, and in some instances on the small sample sizes. To explain whether confounding and interaction did occur, multiple regression and multifactorial analyses of variance were conducted on the data sets; these analyses are shown in Section 4.3.

From the univariate analyses, the influence of domestic pets on symptom reporting and lung function was small in children (Appendix 4.1, Table A.4.1) and non-existent in adults (Table A.4.10). Hobbies which exposed residents to gases, vapours and dusts had a consistent effect on symptom reporting in all females, but the findings on lung function were inconsistent (Tables A.4.2 and A.4.11).

The use of fireplaces had no consistent effects on symptom reporting or lung function in children and adults (Table A.4.3 and A.4.12). Residents of homes with humidifiers generally reported more symptoms, but there were no consistent trends in lung function except for $MF_{25\%}$ and $MF_{50\%}$ in children (Tables A.4.4 and A.4.13). Air conditioning usage was associated with better FEV_1 and slightly more symptoms in children; in adults the use of air conditioning was generally associated with more symptoms (Tables A.4.5 and A.4.14).

The use of hot water heating, and in some cases electric heating systems, seemed to be associated consistently with increased reporting of symptom and decreased lung function in all groups when compared with those who lived in homes with forced air heating (Tables A.4.6 and A.4.15).

The use of gas stoves was associated with lower FEV_1 in white children and increased symptom reporting in black females. In the other groups, the findings were inconsistent (Tables A.4.7 and A.4.16).

Children who lived in crowded homes (>5 persons) had in general slightly more symptoms and decreased lung function (Table A.4.8), but in adults the reverse appeared to be the case (Table A.4.17).

The presence of one smoker in the home was generally associated with slightly more symptom reporting and decreased lung function in white children when compared with those who lived in homes with no smokers (Table A.4.9). The effect of "passive" smoking on the other groups was not consistent in either symptom reporting or lung function (Tables A.4.9 and A.4.18).

4.3 Multiple Regression and Multifactorial Analyses of Variance on Domestic Factors, Symptoms and Lung Function

4.3.1 Stepwise Regression

a. Children

There were some very strong consistent trends in the stepwise analysis for FEV_1 and the exposure variables in children (Table 4.4, Figure 4.2). For white and black males and females the multiple regression was significant ($P<.001$) explaining between 13 to 25% of the variation. The FEV_1 of white boys and girls were separately influenced (negatively) by the use of air conditioners ($P<.01$ for boys, $P<.001$ for girls), the use of hot water heating ($P<.001$) for boys and girls), and the use of gas stoves ($P<.001$ for boys and girls). The FEV_1 of white boys was also influenced separately by crowding ($P<.01$) and

humidifier use ($P<.01$), whereas the FEV_1 of white girls was influenced by the use of electric heating (negatively) and passive smoking (positively).

The FEV_1 of black boys and girls was separately influenced (negatively) by the use of hot water heating ($P<.001$ for both sexes); the use of electric heating ($P<.001$ for both sexes), by the presence of pets ($P<.05$ for both sexes), and positively by domestic crowding ($P<.01$ for both sexes). Black boys' FEV_1 was also influenced separately by the presence of air conditioners in the home ($P<.001$).

For the other two lung function parameters, there were only two significant multiple regressions and these were found for white boys ($P<.05$ for r $MF_{25\%}$, $P<.01$ for r $MF_{50\%}$). The findings for the exposure factors were variable except for electric heating which in general, had negative influences on lung function, and crowding which had very small positive influences on lung function.

The exposure variables were significantly associated with the frequency of cough (Table 4.5) reporting in white boys ($R^2=1.8\%$, $P<.05$) and black girls ($R^2=8.6\%$, $P<.001$). In white boys, the use of gas stoves was separately associated with a decreased reporting of cough ($P<.05$). In black girls, hobbies which exposed them to gases, vapour and dust, and the use of gas stoves separately resulted in the increased reporting of cough ($P<.001$ and $P<.01$ respectively.)

The frequency of phlegm reporting (Table 4.5) was significantly associated with the exposure variables in white girls (R^2=2.7%, $P<.01$) and black boys (R^2=19%, $P<.05$) and girls (R^2=4.0%, $P<.01$). In white and black girls, exposure to the possible irritants from hobbies resulted in increased reporting of phlegm ($P<.01$ for both); in black girls, the number of smokers in the home also had a significant separate negative influence on the reporting of phlegm.

The frequency of wheeze reporting (Table 4.5) was significantly associated with the exposure variables in white boys (R^2=1.8%, $P<.05$), white girls (R^2=1.9%, $P<.05$), and black boys (R^2=2.6%, $P<.01$). There were no significant separate effects of the exposure variable on wheeze in white boys, but in white girls the use of air conditioning ($P<.05$), and having hobbies ($P<.05$) acted separately in causing the increased reporting of wheeze. Black boys who had pets ($P<.05$) and who lived in homes with forced air heating ($P<.01$) and electric heating ($P<.05$) had each exposure factor act separately (negatively) on the reporting of wheeze. In general, the use of electric heating was associated with the increased reporting of wheeze.

The frequency of reporting dyspnea was significantly associated with the exposure variables in white girls (R^2=1.9%, $P<.05$), black boys (R^2=4.8%, $P<.001$), and black girls (R^2=3.7%, $P<.001$). Consistently, the use of electric heating was associated with more dyspnea (not significant) for both boys and girls of both sexes. The number of smokers in the home was associated with less dyspnea in white girls (not significant), in black boys ($P<.001$) and black female ($P<.05$).

b. Adults

There were some consistent trends between the exposure factors and the FEV_1 of adult men and women of both races (Table 4.6). All the multiple regressions were significant for the FEV_1 (white males R^2=4.0%, $P<.05$; white females R^2=1.5%, $P<.01$; black males R^2=22.7%, $P<.01$; and black females R^2=15.0%, $P<.001$). For all groups, the use of hot water heating had a negative effect on lung function ($P<.05$ for white female, $P<.05$ for black males, and $P<.001$ for black females). Domestic crowding had a small positive effect on FEV_1 (for white males (not significant); white females, $P<.05$; and black females, $P<.001$). White and black females who had hobbies which exposed them to gases, vapour and dust had decreased FEV_1 ($P<.05$ for white females).

Other separate effects on FEV_1 were seen in white males from the effect of the number of smokers in the home ($P<.05$); and in white females from the effect of forced air heating ($P<.05$) and electric heating ($P<.05$).

The association between the exposure variables and the $MF_{25\%}$ was smaller than for the FEV_1, the regression being significant for white males (R^2=4.2%, $P<.05$), black males (R^2=19.1%, $P<.05$) and black females (R^2=4.2%, $P<.05$). The only consistent independent effect of any of the exposure variables was seen with the use of hot water heating, but this was evident only in white males ($P<.01$) and black females ($P<.05$). Crowding had a small positive effect in white males (not significant) and black males ($P<.05$).

The $MF_{50\%}$ of white males and black females showed significant associations with the exposure variables (R^2=4.0%, $P<.05$, and R^2=3.5%, $P<.05$ respectively). The use of hot water heaters had a negative independent effect on the $MF_{50\%}$ of white males ($P<.05$), white females (not significant), and black females ($P<.01$). Crowding showed a positive effect on $MF_{50\%}$ for all groups, but only for white males was the effect significant ($P<.01$).

The regression analysis between the exposure variables and cough (Table 4.7) was significant for white females (R^2=1.3%, $P<.05$), and black males (R^2=23.5%, $P<.01$) and females (R^2=5.0%, $P<.01$). The reporting of cough by white and black females was influenced by the use of humidifiers (not significant). The use of hot water heating had a consistent effect resulting in the increased reporting of cough by white males (not significant), black males ($P<.01$), and black females ($P<.001$). Crowding was associated with an increased frequency of reporting cough in white females ($P<.01$).

The regression analysis between the exposure variables and the frequency of reporting phlegm (Table 4.7) was significant in white females (R^2=1.9%, $P<.01$), black males (R^2=32.5%, $P<.01$), and black females R^2=5.3%, $P<.01$). The use of humidifiers was associated with an increased reporting of phlegm in white females ($P<.05$), and black males and females (not significant). The use of forced air heating was associated with the greater reporting of phlegm in white females ($P<.05$), and black males ($P<.001$), and females. Crowding was associated with the reduced reporting of phlegm in white females ($P<.05$), and the use of hot water heating was associated with the increased reporting of

phlegm in black males and females ($P<.001$).

The regression analysis between the exposure variables, and the reporting of wheeze (Table 4.7) were significant for black (R^2=1.5%, ($P<.01$) and white females (R^2=11.9%, $P<.001$). For white females the use of humidifiers separately influenced the increased reporting of wheeze ($P<.01$) whereas crowding resulted in decreased reporting ($P<.05$). In black females, exposure from hobbies was associated with the increased reporting of wheeze ($P<.001$), and the use of hot water heating also was associated with increased reporting ($P<.001$).

The regression analysis between the exposure variables and dyspnea (Table 4.7) were significant for white (R^2=1.4%, $P<.01$) and black females (R^2=5.5%, $P<.01$). There were no logical separate associations between any of the variables and the frequency of reporting dyspnea.

c. Multiple logistic regression analysis on symptom reporting.

There is a limitation in undertaking multiple regression analyses on dichotomous dependent variables. Consequently, the logistic analyses were undertaken to change the variables from binary to continuous. These analyses showed fewer signficant regressions of the five variable model than the linear regression analyses, and fewer of the independent variables acted separately in explaining the symptom prevalence rates. In general the air conditioning, cooking and heating variables were consistent in explaining the symptom rates which was also the case in the linear regression.

TABLES 4.4 - 4.7

STEPWISE MULTIPLE REGRESSION ON SYMPTOMS, LUNG FUNCTION AND EXPOSURE FACTORS

Legend for following four tables:

a. The analysis was conducted on all the exposure variables but only the best 5-variable model is reported. Each model consisted of seven steps which resulted in maximum improvement in the R-square. The symbol (+) indicates the next logical exposure variable in the 5-variable model. The 5-variable model was chosen because most of the other variables above 5 variables were not significant. The model was the best 5-variable model based on the improvement on the R^2 and the contribution of each variable.

b. Exposure codes: HB = hobbies, FP = fireplace, HM = humidifier, AC = air condition, D_2 = dummy variable for forced air heating, D_3 = dummy variable for hot water heating, D_4 = dummy variable for electric heating, CK = cooking fuel, CR = crowding, No.S = number of smokers.

c. P is statistical probability that the regression or the partial regression coefficient is significant at * (5% level), ** (1% level), *** (0.1% level), NS = not significant.

d. W = White, M = Male
B = Black, F = Female

e. ( ) no of persons in group.

f. In these analyses, the coding was as follows: (Figure 4.2)
for PEJ, HB, FP, HM, AC: 1 = yes, 2 = no.
for CK: 1 = electricity, 2 = gas
for D_2, D_3, D_4: 1 = other heating, 2 = heating variable
for symptoms: 1 = yes, 2 = no.

FIGURE 4.2

INTERPRETATION OF THE SIGN ON THE REGRESSION COEFFICIENT

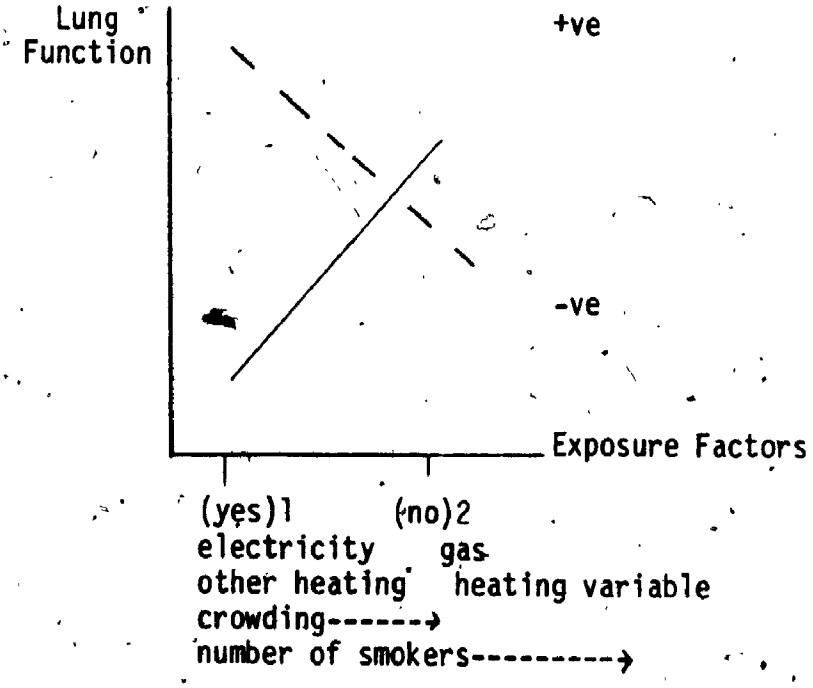


TABLE 4.4

REGRESSION OF LUNG FUNCTION ON EXPOSURE FACTORS IN CHILDREN

	PET	HB	FP	HM	AC	D2	D3	D4	CK	CR	NO.S	R^2	P
r FEV1													
WM (729)				.076**	-.146**		-.227***	+	-.148***	-.025**		24.8	***
WF (628)					-.198***		-.209***	-.089**	-.075***	+	.030**	23.7	***
BM (562)	-.061*				-.082***		-.263***	-.100***	+	.010**		13.3	***
BF (494)	-.042*		-.046*				-.325***	-.084***	+	.014**		18.4	***
r MF25%													
WM	.131**	-.070			-.062	-.067		-.094	+			1.8	*
WF		.066	.055		.046			-.070		.017	+	1.4	NS
BM				.094		.056			-.045	.012	-.017	0.9	NS
BF		.266		.134		.074	+	.055		.007		1.5	NS
r MF50%													
WM	.308	-.115*			-.056	-.151**	-.162*	+				2.0	**
WF			.047		.057		+	-.048	.024	.019		0.9	NS
BM			.106	.171		-.079			+	.028*	-.034	1.7	NS
BF		.234		.280*		.106	+	-.067	.052			1.7	NS

TABLE 4.5

REGRESSION OF SYMPTOMS ON EXPOSURE FACTORS IN CHILDREN

	PETS	HB	FP	HM	AC	D2	D3	D4	CK	CR	NO.S	R_2	P
Cough													
WM	.012					-.009	+		.028**	-.001	-.008	1.8	*
WF				.032*	.021	.026	.021	.025		+		1.6	NS
BM		-.026	.031		.013			-.024	+	-.005*		1.8	NS
BF	.029	.388***				-.025	-.043		-.041	+		8.6	***
PHLEGM													
WM				-.011		-.015	-.028	-.024	.009			.7	NS
WF		.040**	.010	-.009	-.013					+	-.004	2.7	**
BM	-.015	-.023	-.017			-.021	-.045				+	1.9	*
BF	.015	.135**	-.018			.016				+	-.012***	4.0	**
Wheeze													
WM			.039	.065	.040	.042			.046	+		1.8	*
WF		.089*			.061*		+	-.025	.037		-.007	1.9	*
BM	.057*	.060					-.130**	-.097*		+	-.014	2.6	**
BF	-.044*		.047		.033		+	-.037		.004		1.5	NS
Dyspnea													
WM					-.024	-.075**	-.067*	-.059	-.018	+		1.2	NS
WF		-.062			-.081**			-.038	+	.012	.012	1.9	*
BM	-.022		-.043			-.040	+	-.050			.050***	4.3	***
BF		-.486***					.111	-.059	-.028	+	.036*	3.7	***

TABLE 4.6

REGRESSION OF LUNG FUNCTION ON EXPOSURE FACTORS IN ADULTS

	PET	HB	FP	HM	AC	D2	D3	D4	CK	CR	NO.S	R^2	P
r FEV1													
WM (315)	-.089			-.106			-.049		+	.020	-.070*	4.0	*
WF (962)		-.055*				-.061*	-.073*	-.118*		.011*		1.5	**
BM (59)	.093				.137*		-.223*	.235	.078		+	22.7	**
BF (325)		-.181	-.078*				-.209***		+	.022***	.017	15.0	***
r MF25%													
WM			-.077	-.177		-.209	-.309**		+	.034		4.2	*
WF				-.017	-.020			-.095	-.031	+	-.024	0.5	NS
BM		-.261				.248	+		-.124	.070*	.111	19.1	*
BF		.378		.212		-.087	-.239*		.120			4.2	*
r MF50%													
WM				-.232		-.464*	-.453*	-.243		.100**	+	4.0	*
WF		-.117			-.061		-.052	+		.019	-.084	0.6	NS
BM		-.990	.444		.353			.471		.052		10.9	NS
BF	.166		-.169				-.467**		.064	.009	+	3.5	*

TABLE 4.7

REGRESSION OF SYMPTOMS ON EXPOSURE FACTORS IN ADULTS

	PETS	HB	FP	HM	AC	D2	D3	D4	EK	CR	NO.S	R^2	P
Cough													
WM					+	-.091	+.059	-.109		-.010	.041**	2.6	NS
WF		.026	-.025	.022			+		-.014	.013**		1.3	*
BM	-.103		-.086			.072	-.347***			-.012	+	23.5	**
BF	.014			.044	+		-.087***	-.044	.010			5.0	**
Phlegm													
WM			-.041				+	.062	.052	.010	-.024	2.3	NS
WF	.013		-.029*	.039*		-.026*	+			.016*		1.9	**
BM				.267	-.085	-.190*	-.652***		-.055		+	32.5	**
BF				.041		-.020	-.096***	-.050		-.082	+	5.3	**
Wheeze													
WM	-.022	.100			.021		+	-.043		.010		1.4	NS
WF				.082**	.035	.033	+			.010*	-.022	1.5	**
BM		-.185			-.139	.108	-.196				.075	12.2	NS
BF		.722***	-.119*	.151		-.038	-.209***				+	11.9	***
Dyspnea													
WM						.050*	-.081	-.137	+	-.016	-.030	2.9	NS
WF	.120		.090*			-.028	-.149		+		.038	1.4	**
BM			.081		.114		.156			-.011	.031	8.5	NS
BF	-.114*	-.897***	.115	-.214						+	-.027	5.5	**

c. Summary

The multiple regression analyses showed that the exposure variables explained more of the variation in the FEV_1 than the other two lung function variables or the four symptom variables. In general, the influence of domestic pets and the use of fireplaces and humidifiers was small. The presence of one or more smokers in the home had very little separate influence on symptoms and lung function, whereas the use of hot water heating and electric heating had mild negative influences on lung function and symptom reporting. In children the use of air conditioners resulted in better lung function and generally fewer symptoms. These trends were not as clearly evident in the univariate analyses. In white children the influence of the use of gas stoves was evident in the multiple regression (which was also seen in the individual univariate analyses). The use of the multiple logistic regression analysis on the symptom variables did not result in any improvement in the quality of the results.

4.3.2 Multifactorial Analyses of Variance with Two Interaction Variables

These analyses were conducted with all the exposure variables except pets since the earlier analyses showed inconsistent weak effects of this variable. The multifactorial analyses were conducted for: children and adults separately, black and white, males and females, the three lung function parameters, and the four symptoms. These analyses assisted in the determination whether the exposure variables were acting together in an additive or multiplicative way.

a. Children

The significant interactions between pairs of the exposure variables are shown in matrix tables in Appendices 4.2.1 to 4.2.7. The most

consistent interactions were found for r FEV_1, and to a lesser extent, the four symptom variables.

i. $\underline{FEV_1}$

For r FEV_1, the significant interactions were observed mainly between (a) the air conditioning variable and the heating, cooking fuel, crowding and number of smokers variables, (b) the heating variable and the cooking fuel, crowding, and number of smokers variables, and (c) to a lesser extent between the cooking fuel variable and crowding, and the number of smokers (mainly in white girls) variables. There was interaction between hobbies and the heating and number of smokers variables, respectively, in black boys only. Interactions between humidifier use and the other variables, and between fireplace use and the other variables were virtually absent.

Because of the consistent significant findings for the r FEV_1, summaries of all of the mean values for r FEV_1 for children are shown for all the interactions except humidifiers and fireplaces, in Tables 4.8 to 4.15.

Table 4.8 shows the mean r FEV_1 for the interactions between air conditioning use and the five other exposure variables. The reduction in FEV_1 related to gas stove use compared with electric stove use was more marked in homes without air conditioning for white boys and girls. The effect of air conditioning use and the degree of passive smoking on FEV_1 was small. Air conditioning use and hobbies had some small but

non-significant interactions. In white children, the interactions between air conditioning use and the heating variables were significant, but in black children they were inconsistent. In white children, there appeared to be interaction between crowding and air conditioning usage.

Table 4.9 shows the interaction between the number of smokers in the home and four other exposure variables. There were no consistent trends between the number of smokers in the home and domestic crowding. There was no consistent interaction between the number of smokers and the type of cooking stove used. The presence of one smoker in the home showed consistent interactions with the use of hot water heating in white girls and black boys and girls, and also with hobbies which exposed white boys and girls and black boys to gases, vapours and dusts.

Table 4.10 shows the interactions between heating and three other exposure variables. The interaction between hot water heating and exposure to hobbies was inconsistent, but in white children the interaction between the heating variables and gas stoves was generally consistent. In general there appeared to be a consistent interaction between the heating variables and domestic crowding in white girls, black boys and black girls.

Table 4.11 shows the interactions between cooking fuel use and hobbies and crowding respectively, and between hobbies and crowding individually. The use of gas stoves showed some interaction with hobbies and domestic crowding among white children. In black children the trends were usually reversed. Hobbies and crowding together among white children also showed some reduction in FEV_1.

ii. $\underline{MF_{25\%}}$ and $\underline{MF_{50\%}}$

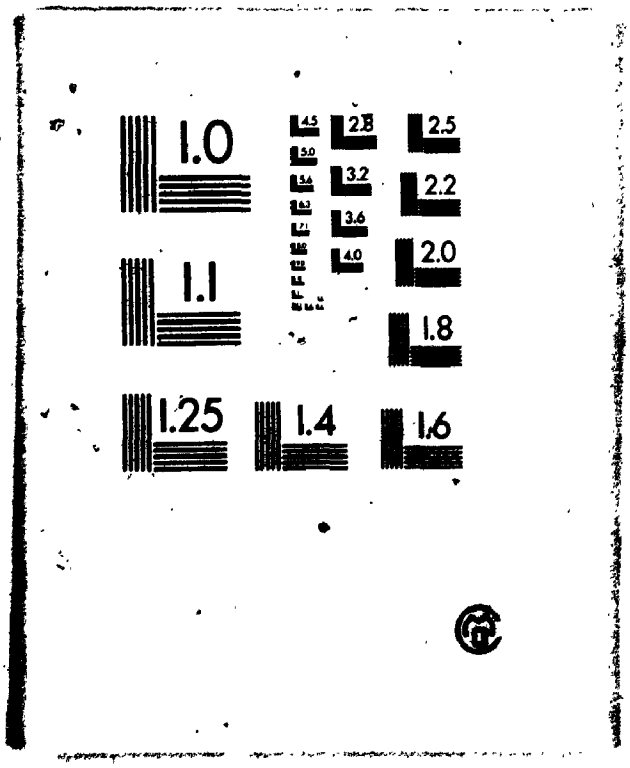
There were very few significant interactions between the exposure variables and the other two lung function parameters (Appendices 4.2.2 and 4.2.3), and consequently these will not be discussed further.

iii. Cough

There were significant interactions between crowding and the use of air conditioning and the number of smokers in the home (respectively) in girls. In black boys there was more cough reported by those who lived in crowded homes without air conditionjng (Appendix 4.2.4). In black girls, there was significant interactions between hobbies, air conditioning, heating, crowding and the number of smokers.

iv. Phlegm

Among white girls, there was more phlegm in those who had hobbies and were without air conditioning, and who had hot water heating respectively. Among black boys there was more phlegm in those whose homes had no air conditioning but had hot water heating, and the same occurred in crowded homes without air conditioning. Among black girls, there were many interactions between hobbies and some of the other exposure variables, but the numbers in the groups were too small to show consistent trends (Appendix 4.2.5).


1.0
1.1
1.25
1.4
1.6
2.8
3.2
3.6
4.0
2.5
2.2
2.0
1.8

v. Wheeze

Among white girls, there was more wheeze in those who had no air conditioning and who lived in homes with more than one smoker. The same effect was seen among black girls. Black girls who lived in homes with hot water heating and which were crowded had more frequent reporting of wheeze. (Appendix 4.2.6)

vi. Dyspnea

Amongst white girls, there was more dyspnea in those who had hobbies and respectively lived in homes with air conditioning, lived in crowded homes, and lived in homes with one or more smokers. Amongst white boys there was interaction between crowding and air conditioning (Appendix 4.2.7). In black girls there was interaction between hobbies, air conditioning, heating and cooking fuel.

b. Adults

There were fewer interactions between the exposure variables in adult as compared with children (Appendices 4.2.1 to 4.2.7). Because of the small number of black adults, no comments will be made on the outcome of their analyses. Also, because of the very few interactions with fireplaces and humidifiers, these will be excluded from further comments.

i. $\underline{FEV_1}$

There were no consistent interactions between the use of air conditioners and any of the other five exposure variables (Table 4.12). The interactions between the number of smokers in the home with crowding, cooking fuel used, and hobbies respectively showed no trends. There was some small interaction between the use of hot water heating and the number of smokers in the home (not significant) (Table 4.13).

The use of hot water heating and electric heating together with hobbies showed a small reduction on FEV_1 of white men (Table 4.14). There were consistent trends with heating and the type of cooking fuel used, and there was a small reduction in FEV_1 from the use of electric heating with domestic crowding.

There were no significant or consistent interactions between the type of cooking fuel used and hobbies or crowding (Table 4.15). Neither was there any interaction between hobbies and crowding on the FEV_1 of adults (Table 4.15).

ii. $\underline{MF_{25\%} \text{ and } MF_{50\%}}$

There were very few significant interactions between the exposure variables for the other two lung function parameters in adults (Appendices 4.2.2 and 4.2.3). Consequently there would be no further discussion on these.

iii. Cough

There were no consistent trends for cough in white men and women. For black men and women, more cough was reported by those who used air conditioning and lived in homes with smokers. Also in black men and women, there was more cough reported by those who lived in crowded homes with hot water heating. (Appendix 4.2.4.).

iv. Phlegm

White males who had air conditioning and hobbies reported more phlegm than those who did not have air conditioning and hobbies. Black females who had air conditioning in their homes reported more phlegm when there were smokers in the home and when the homes were crowded respectively. Black females who lived in homes with hot water heating reported more phlegm when the homes were crowded and also when gas stoves were used (respectively) (Appendix 4.2.5).

v. Wheeze

White males who lived in homes with gas stoves and with air conditioning, reported more wheeze; they also reported more wheeze if they lived in homes with gas stoves and with smokers. White males who had hobbies reported more wheeze when they had hot water heating and they lived in crowded homes (respectively) (Appendix 4.2.6).

vi. Dyspnea

White males who had air conditioning reported more dyspnea when they also had hobbies. This group also reported more dyspnea when they lived in homes with gas stoves and hot water heating. More dyspnea also resulted among white males when they lived in crowded homes with air conditioning (Appendix 4.2.7).

c. Summary

The findings from the multifactorial analyses of variance showed that six of the exposure variables consistently acted together in pairs to result in lower FEV_1 and higher frequencies of symptom reporting. The most consistent effects were observed for hobbies, air conditioning use, heating, cooking fuel, crowding, and the number of smokers in the home.

In the multiple regression analyses, crowding and the number of smokers in the home had independent small positive effects on respiratory health; in the multifactorial analyses of variance each of these two variables acted together with each of the other variables to produce generally small negative effects on respiratory health.

4.3.3 Multifactorial Analyses of Variance with Three Interaction Variables.

Two exposure variables that consistently showed significant interactions were used in a 3-way interaction model with all the other variables

TABLES 4.8 to 4.15

MULTIFACTORIAL ANALYSES
OF VARIANCE BETWEEN THE EXPOSURE VARIABLES AND FEV_1

Legend for tables that follow:

a. Exposure variables are:

AC = air conditioning
CK = cooking fuel
CR = crowding
FP = fireplace
HB = hobbies
HT = heating
No. S = number of smokers
FA = forced air
HW = hot water
EL = electric

b. Subject variables are:

BF = black females
BM = black males
WF = white females
WM = white males

c. Level of significance:

\- = not significant
* = $P < .05$
** = $P < .01$
*** = $p < .001$

TABLE 4.8 MEAN rFEV$_1$ FOR PAIRS OF EXPOSURE VARIABLES IN CHILDREN

		AIR CONDITION							
		NO				YES			
COOKING FUEL		ELEC	GAS			ELEC	GAS		
WM	u	.167	.049			.390	.200		
	n	250	178			211	90		
WF	u	*.138	.114			.416	.276		
	n	246	153			165	64		
BM	u	.276	.355			.384	380		
	n	262	195			70	35		
BF	u	.342	.409			.289	.384		
	n	219	182			59	34		
NO. OF SMOKERS		0	1	2		0	1	2	
WM	u	*.077	.137	.137		.375	.295	.331	
	n	133	152	143		110	113	78	
WF	u	*.098	.084	.190		.389	.381	.355	
	n	110	135	154		85	79	65	
BM	u	.320	.288	.327		.388	.399	.351	
	n	169	163	125		49	32	24	
BF	u	*.367	.396	.349		.258	.382	.391	
	n	143	139	119		45	26	22	
BOBBIES		NO	YES			NO	YES		
WM	u	.124	.103			.345	.286		
	n	300	128			243	58		
WF	u	.137	.069			.391	.208		
	n	354	45			211	18		
BM	u	.312	.281			.386	.365		
	n	426	31			90	15		
BF	u	.372	.383			.322	.453		
	n	395	6			92	1		
HEATING		FA	HW	EL		FA	HW	EL	
WM	u	***.161	.022	.134		.410	.009	.396	
	n	158	159	44		183	51	34	
WF	u	***.181	.034	.110		.443	.078	.398	
	n	147	145	51		143	29	30	
BM	u	.368	.022	.214		.393	.291	.314	
	n	170	30	46		41	5	18	
BF	u	.419	.074	.343		.369	.008	.226	
	n	130	23	58		35	7	22	
CROWDING		1.5	3.5	5.5	6.5	1.5	3.5	5.5	6.5
WM	u	*.078	.198	.095	.073	.477	.328	.367	.202
	n	4	121	191	112	8	98	152	43
WF	u	*.156	.089	.143	.144	.470	.374	.407	.219
	n	5	106	187	101	2	88	117	22
BM	u	.321	.254	.287	.338	.319	.363	.314	.429
	n	5	73	126	253	2	19	29	55
BF	u	.287	.271	.381	.388	0	.271	.299	.389
	n	5	43	103	250	0	27	32	34

u = mean n = no subjects

TABLE 4.9 MEAN rFEV$_1$ FOR PAIRS OF EXPOSURE VARIABLES IN CHILDREN

NO. OF SMOKERS

		0				1				≥2			
CROWDING		1.5	3.5	5.5	6.5	1.5	3.5	5.5	6.5	1.5	3.5	5.5	6.5
WM	u	.202	.274	.225	.031	.507	.267	.183	.122	.691	.193	.245	.143
	n	7	93	104	39	4	84	130	47	1	42	109	69
WF	u	.083	.230	.266	.083	.463	.197	.197	.149	-	.235	.272	.191
	n	4	72	93	26	3	76	104	31	0	46	107	66
BM	u	.337	.345	.311	.347	.223	.183	.277	.355	-	.240	.275	.359
	n	6	48	70	94	1	29	57	108	0	15	28	106
BF	u	.268	.236	.320	.409	.301	.338	.396	.401	-	.329	.374	.352
	n	2	45	52	89	3	11	51	100	0	14	32	95

COOKING FUEL		EL	GAS	EL	GAS	EL	GAS
WM	u	.294	.050	.260	.093	.248	.150
	n	161	82	175	90	125	96
WF	u	.244	.188	.256	.076	.249	.221
	n	128	67	140	74	143	76
BM	u	.324	.350	.271	.350	.298	.390
	n	126	92	110	85	96	53
BF	u	.296	.406	.359	.433	.346	.367
	n	109	79	88	77	81	60

HEATING		FA	HW	EL	FA	HW	EL	FA	HW	EL
WM	u	.293	-.011	.283	.299	.034	.241	.292	.034	.213
	n	120	68	30	120	80	23	101	62	25
WF	u	.331	.030	.125	.312	-.032	.312	.284	.124	.226
	n	98	46	29	99	64	23	93	64	31
BM	u	.369	.286	.222	.382	-.105	.180	.367	.004	.362
	n	91	12	25	60	13	23	60	10	16
BF	u	.381	.121	.182	.446	.065	.417	.392	-.006	.358
	n	58	8	31	65	13	29	50	9	20

HOBBIES		NO	YES	NO	YES	NO	YES
WM	u	.221	.185	.218	.161	.232	.132
	n	180	63	200	65	163	58
WF	u	.229	.173	.205	.089	.260	.084
	n	179	16	193	21	193	26
BM	u	.330	.375	.317	.149	.329	.360
	n	193	25	182	13	141	8
BF	u	.341	.417	.394	.404	.356	.291
	n	185	3	162	3	140	1

TABLE 4.10 MEAN $rFEV_1$ FOR PAIRS OF EXPOSURE VARIABLES IN CHILDREN

		HEATING					
		ELECTRIC		FORCED AIR		HOT WATER	
HOBBIES		NO	YES	NO	YES	NO	YES
WM	u	.255	.232	.303	.260	.015	.027
	n	54	24	275	66	140	70
WF	u	.235	.112	.321	.163	.042	.039
	n	69	12	269	21	147	27
BM	u	.261	.056	.364	.457	.073	-.072
	n	58	6	192	19	32	3
BF	u	.310	-.354	.409	.387	.058	-
	n	79	1	169	4	30	0
COOKING FUEL		ELEC	GAS	ELEC	GAS	ELEC	GAS
WM *	u	***.259	.140	.393	.135	.029	.002
	n	71	7	211	130	130	80
WF	u	**.198	.297	.365	.201	.046	.033
	n	66	15	192	98	114	60
BM	u	***.187	.384	.363	.382	.006	.380
	n	46	18	113	98	29	6
BF	u	**.279	.436	.398	.418	.021	.399
	n	64	16	85	88	27	3

CROWDING		1.5	3.5	5.5	6.5	1.5	3.5	5.5	6.5	1.5	3.5	5.5	6.5
WM	u	.691	.247	.264	.190	.390	.355	.286	.187	.004	.040	.013	.006
	n	1	23	37	17	9	108	168	56	1	62	92	55
WF	u	**.458	.233	.256	.109	.310	.343	.331	.172	-	.018	.042	.075
	n	1	20	40	20	5	91	149	45	0	54	84	36
BM	u	* -	.136	.180	.333	.268	.341	.386	.378	.450	-.139	.091	.118
	n	0	10	25	29	2	36	53	120	1	8	11	15
BF	u	*** -	.074	.200	.423	.363	.394	.398	.420	-	-.145	.218	0.62
	n	0	13	20	47	4	19	59	91	0	5	6	.19

TABLE 4.11 MEAN rFEV$_1$ FOR PAIRS OF EXPOSURE VARIABLES FOR CHILDREN

		COOKING FUEL			
		ELECTRICITY		GAS	
HOBBIES		NO	YES	NO	YES
WM	u	.291	.204	.105	.087
	n	344	117	199	69
WF	u	.264	.124	.171	.075
	n	368	43	197	20
BM	u	.299	.306	.362	.315
	n	299	33	217	13
BF	u	.330	.394	.405	.392
	n	275	3	212	4

		COOKING FUEL							
		ELECTRICITY				GAS			
CROWDING		1.5	3.5	5.5	6.5	1.5	3.5	5.5	6.5
WM	u	.547	.321	.269	.156	.061	.137	.106	.056
	n	7	141	231	5	5	78	112	73
WF	u	.562	.294	.255	.157	.120	.084	.222	.158
	n	2	124	207	78	5	70	97	45
BM	u	.335	.229	.251	.342	.232	.342	.349	.371
	n	6	53	91	182	1	39	64	126
BF	u	.182	.196	.340	.364	.358	.384	.394	.415
	n	2	42	81	153	3	28	54	131

		No Hobbies				Yes Hobbies			
CROWDING		1.5	3.5	5.5	6.5	1.5	3.5	5.5	6.5
WM	u	.324	.254	.242	.128	.572	.259	.143	.052
	n	11	165	251	116	1	54	92	39
WF	u	.200	.232	.257	.166	.521	.069	.115	.110
	n	6	178	277	104	1	16	27	19
BM	u	.337	.270	.287	.359	.223	.329	.334	.282
	n	6	81	140	289	1	11	15	19
BF	u	.288	.266	.360	.387	-	.380	.403	-
	n	5	67	131	284	0	3	4	0

TABLE 4.12 MEAN rFEV$_1$ FOR PAIRS OF EXPOSURE VARIABLES IN ADULTS

		AIR CONDITION					
		NO			YES		
COOKING FUEL		ELEC	GAS		ELEC	GAS	
WM	u	-.034	.012		-.007	.029	
	n	111	75		87	42	
WF	u	-.001	-.013		.003	-.005	
	n	314	251		282	115	
BM	u	-	-		-	-	
	n	-	-		-	-	
BF	u	.098	.125		.046	.102	
	n	145	128		30	22	
NO. OF SMOKERS		0	1	2	0	1	2
WM	u	.030	-.085	-.203	-.013	.034	.084
	n	132	35	19	92	26	11
WF	u	-.014	.007	-.015	-.010	.001	.044
	n	313	180	66	227	117	53
BM	u	-	-	-	-	-	-
	n	-	-	-	-	-	-
BF	u	.080	.148	.121	.088	.074	-.042
	n	126	86	61	33	13	6
HOBBIES		NO	YES		NO	YES	
WM	u	-.038	.099		.021	-.125	
	n	155	31		114	15	
WF	u	-.015	.055		-.002	.026	
	n	497	68		356	41	
BM	u	-	-		-	-	
	n	-	-		-	-	
BF	u	.107	.374		.069	-	
	n	269	4		52	0	
HEATING		FA	HW	EL	FA	HW	EL
WM	u	-.072	-.019	.117	.060	-.099	-.123
	n	54	94	18	80	29	9
WF	u	-.012	-.009	-.050	.004	.020	-.049
	n	217	225	49	254	82	29
BM	u	-	-	-	-	-	-
	n	-	-	-	-	-	-
BF	u	.111	-.094	.117	.156	-.095	-.011
	n	78	22	14	23	8	6
CROWDING		1.5	3.5	5.5	6.5	1.5	3.5	5.5	6.5
WM	u	-.067	-.066	.025	.019	-.024	.001	.034	.032
	n	56	71	39	20	31	61	32	5
WF	u	-.018	.009	-.019	-.013	-.037	.001	.060	.073
	n	174	229	128	34	143	167	75	12
BM	u	-	-	-	-	-	-	-	-
	n	-	-	-	-	-	-	-	-
BF	u	-.047	.110	.090	.184	.078	.090	.042	.073
	n	38	69	65	101	10	16	16	10

TABLE 4.13 MEAN rFEV$_1$ FOR PAIRS OF EXPOSURE VARIABLES IN ADULTS

NO. OF SMOKERS

		0				1				>2+			
CROWDING		1.5	3.5	5.5	6.5	1.5	3.5	5.5	6.5	1.5	3.5	5.5	6.5
WM	u	-.004	-.005	.005	.084	-.296	.107	-.028	-.032	-	-.162	-.059	-.016
	n	73	91	50	10	14	25	18	4	0	16	3	11
WF	u	-.039	-.001	.025	.053	.007	.030	-.041	-.029	.006	-.040	.067	-.008
	n	237	206	81	16	75	144	75	9	5	46	47	21
BM	u	-	-	-	-	-	-	-	-	-	-	-	-
	n	-	-	-	-	-	-	-	-	-	-	-	-
BF	u	-.040	.120	.093	.163	.092	.090	.058	.244	-	.076	.086	.121
	n	41	48	35	35	7	32	24	36	0	5	22	40

		0		1		>2+	
COOKING		ELEC	GAS	ELEC	GAS	ELEC	GAS
WM	u	-.005	.044	-.005	-.080	-.231	.036
	n	146	78	37	24	15	15
WF	u	-.008	-.019	.001	.011	.047	-.035
	n	344	196	185	118	67	52
BM	u	-	-	-	-	-	-
	n	-	-	-	-	-	-
BF	u	.101	.058	.095	.195	.043	.161
	n	88	71	56	43	31	36

		0			1			>2+		
HEATING		FA	HW	ELEC	FA	HW	ELEC	FA	HW	ELEC
WM	u	.004	.015	-.003	-.004	-.134	.301	.066	-.227	-.281
	n	92	87	10	31	23	4	11	13	2
WF	u	-.023	-.010	-.028	.025	-.019	-.045	.022	-.002	-.139
	n	278	156	37	130	114	30	63	37	11
BM	u	-	-	-	-	-	-	-	-	-
	n	-	-	-	-	-	-	-	-	-
BF	u	.106	-.058	.051	.152	.011	.059	.104	-.288	.175
	n	54	11	10	35	11	6	12	8	4

		0		1		>2+	
HOBBIES		NO	YES	NO	YES	NO	YES
WM	u	.014	.003	-.060	-.167	.110	-.018
	n	189	35	64	7	26	4
WF	u	-.015	.013	-.005	.073	.001	.087
	n	484	56	264	39	105	14
BM	u	.055	.072	-.069	-	.190	-
	n	41	4	7	0	7	0
BF	u	.079	.500	.135	.311	.103	.371
	n	158	1	96	2	66	1

TABLE 4.14

MEAN $rFEV_1$ FOR PAIRS OF EXPOSURE VARIABLES IN ADULTS

		HEATING					
		ELECTRIC		FORCED AIR		HOT WATER	
HOBBIES		NO	YES	NO	YES	NO	YES
WM	u	.049	-.063	-.003	.083	-.036	-.049
	n	24	3	118	16	101	22
WF	u	-.067	.033	-.008	.041	-.019	.039
	n	65	13	423	48	268	39
BM	u	-	-	-	-	-	-
	n	-	-	-	-	-	-
BF	u	-.078	-	.120	.258	-.094	-
	n	20	0	100	1	30	0

COOKING FUEL		ELEC	GAS	ELEC	GAS	ELEC	GAS
WM	u	.026	.100	.049	-.053	-.098	.077
	n	23	4	79	55	81	42
WF	u	-.040	-.118	.018	-.040	-.017	-.004
	n	68	10	300	171	185	122
BM	u	-	-	-	-	-	-
	n	-	-	-	-	-	-
BF	u	.057	.267	.114	.133	-.060	-.315
	n	18	2	61	40	26	4

CROWDING		1.5	3.5	5.5	6.5	1.5	3.5	5.5	6.5	1.5	3.5	5.5	6.5
WM	u	.312	-.003	-.035	-.139	-.078	.022	.081	.013	-.050	-.051	-.049	.099
	n	5	13	7	2	36	57	29	12	32	54	27	10
WF	u	-.009	-.058	-.102	.030	-.043	.020	.015	.011	-.021	-.004	-.014	-.024
	n	19	31	21	7	164	183	107	17	88	147	54	18
BM	u	-	-	-	-	-	-	-	-	-	-	-	-
	n	-	-	-	-	-	-	-	-	-	-	-	-
BF	u	.019	.172	-.124	.129	.031	.088	.111	.230	-.032	-.032	-.256	-.015
	n	2	5	4	9	17	30	28	26	4	8	9	9

TABLE 4.15 MEAN rFEV$_1$ FOR PAIRS OF EXPOSURE VARIABLES IN ADULTS

		COOKING FUEL			
		ELECTRICITY		GAS	
HOBBIES		NO	YES	NO	YES
WM	u	-.015	-.062	-.009	.177
	n	169	29	100	17
WF	u	-.002	.026	-.022	.074
	n	528	68	325	41
BM	u	-	-	-	-
	n	-	-	-	-
BF	u	.086	.311	.117	.435
	n	173	2	148	2

		COOKING FUEL							
		ELECTRICITY				GAS			
CROWDING		1.5	3.5	5.5	6.5	1.5	3.5	5.5	6.5
WM	u	-.032	-.028	-.033	.105	-.088	.050	.114	-.055
	n	56	88	41	12	31	43	30	13
WF	u	-.036	.018	.022	.030	-.008	-.014	-.007	-.020
	n	204	242	123	27	113	154	80	19
BM	u	-	-	-	-	-	-	-	-
	n	-	-	-	-	-	-	-	-
BF	u	.008	.111	.034	.160	-.037	.101	.140	.190
	n	26	46	46	57	22	39	35	54

		NO HOBBIES				YES HOBBIES			
CROWDING		1.5	3.5	5.5	6.5	1.5	3.5	5.5	6.5
WM	u	-.063	.013	-.005	.001	.024	-.101	.182	.166
	n	76	113	58	22	11	19	13	3
WF	u	-.031	-.001	.008	-.006	.027	.052	.024	.112
	n	291	342	180	40	26	54	23	6
BM	u	-	-	-	-	-	-	-	-
	n	-	-	-	-	-	-	-	-
BF	u	-.021	.106	.073	.171	0	0	.379	.367
	n	48	85	79	109	0	0	2	2

individually. In these analyses, only a total of six of the nine variables have been included (pets, humidifier use and fireplace use were excluded).

The two exposure variables which were compared with the others were air conditioning and cooking fuel. Because of the trends seen with the crowding variable, rather than use four crowding groups, for the following analyses, two groups were used with Group 1 having less than five residents and Group 2 having five or more residents.

a. Children

Table 4.16 shows that when two variables (AC*CK) were included in the multivariate analyses, there were very significant interactions with the third variable for r FEV_1. The mean r FEV_1 was computed for all the three variable models (Tabel 4.18) and in general they revealed that (i) white children who had hobbies and lived in homes without air conditioning, but had gas stoves had the lowest mean r FEV_1 when compared with those children who had no hobbies and lived in air conditioned homes with electric stoves. The difference between the mean FEV_1 for both groups was .329 liters for boys and .386 liters for girls. If the individual reduction in FEV_1 from the univariate analyses (Appendix 4.1, Tables A.4.2-A.4.9) 4.2-4.9) were summed, then the expected reduction in FEV_1 for these exposure variables would be .44 and .45 liters for white boys and girls respectively (Table 4.20).

ii. White children, who lived in homes without air conditioning, but had gas stoves and hot water heating, had the lowest mean r FEV_1 when compared with their counterparts living in homes with air conditioning, electric stoves and forced air heating. The difference in the mean FEV_1, from the interaction was .468 liters for boys and .489 liters for girls. By addition of individual exposure effects from Tables A.4.2-A.4.9, the mean FEV_1 reduction was .65 and .60 liters respectively (Table 4.20).

iii. White children who lived in homes without air conditioning and which had gas stoves and one smoker had slightly lower mean FEV_1 when compared with those who lived in homes with air conditioning, electric stoves and zero smokers. The difference in the mean FEV_1 between these groups was .351 liters and .336 liters for boys and girls respectively. By addition of the individual exposure effects on FEV_1 from Tables A.4.2-A.4.9, the mean FEV_1 reduction was 0.39 liters and 0.36 liters respectively (Table 4.20).

iv. White children who lived in crowded homes without air conditioning but with gas stoves had significantly lower FEV_1 compared to their counterparts in less crowded homes with air conditioning and with electric stoves. The difference in the mean FEV_1 between these groups was .397 liters for boys and .274 liters for girls. By addition of the individual exposure effects of FEV_1 from Tables A.4.2-A.4.9, the mean FEV_1 reduction was .50 liters and .38 liters respectively (Table 4.20).

In black children, although the 3-way interactions were significant, the reduction in FEV_1 was small in each case for boys, and in the opposite direction for girls.

For all children, there were very few significant 3-way interactions between the exposure variables on the frequency of reporting symptoms. There were no consistent trends for any one symptom, and within symptoms, there were no consistent differences in the frequency of reporting between the 3-way groups.

b. Adults

There were fewer significant 3-way interactions in adults (Table 4.16). In white males, there was interaction between air conditioning, cooking fuel and heating, but the mean FEV_1 between the exposure groups was not consistent with the exposure (Table 4.19). In white females, the interaction between air conditioning, cooking fuel and hobbies was significant (Table 4.16), but the effect on the FEV_1 was not consistent with the exposure (Table 4.19). In white females, the interaction of air conditioning, cooking and crowding resulted in a lower mean FEV_1 of 0.02 liters. Black males were not considered in the analyses, but for black females the 3-way interaction with air conditioning, cooking and heating, and with air conditioning, cooking and crowding were significant (Table 4.16), but the number of persons in some of the groups were too small for meaningful comparisons (Table 4.19).

In white adults there was only one significant interaction for the symptoms (white males-dyspnea). In black females, there were some significant interactions for cough and phlegm, but the numbers in some of the groups were too small to draw conclusions.

c. Summary

The three way interactions between the exposure variables were most consistent among white boys and girls, and less evident in black boys and girls, and adults. The exposure variables with the strongest effects on FEV_1 were air conditioning, hobbies, heating, cooking and crowding. Smokers in the home had a very weak effect on non-smokers. The effects of the exposure variables were very small and inconsistent on the symptom variables.

4.3.4 Multifactorial Analyses of Variance with Four Interaction Variables

The four exposure variables which showed consistently the main effects were used in the 4-way analysis. The variables included hobbies, air conditioning, heating and cooking.

a. Children

There was significant interaction between the four variables on r FEV_1 in all children (Table 4.16). Among white boys and girls, those who had hobbies and lived in homes without air conditioning, but had gas stoves

and hot water heating had lower FEV_1 (.387 liters for boys and .461 liters for girls) than those who had no hobbies and who lived in air conditioned homes with electric stoves and forced air heating (Table 4.20). By adding the individual reduction contributed by each exposure variable from the unvariate analyses (Tables A.4.2 to A.4.9), the reduction was .71 liters and .72 liters respectively. In black children the numbers in the groups were too small to make any meaningful deductions.

The 4-way interaction on the symptoms were significant for white girls for phlegm and dyspnea reporting, and black boys for wheeze reporting, and in black girls for cough, phlegm and dyspnea reporting. A review of the frequency data for these interactions showed no consistent trends.

b. Adults

The 4-way interaction in adults showed some significant interactions for black adult females for FEV_1, cough, phlegm and wheeze, but because the number of subjects in some of the exposure groups were so small, no further analyses were made.

c. Summary

The four-way interactions were consistent only for white boys and girls and only on the FEV_1. The reduction in FEV_1 was substantial in those who had hobbies and lived in homes with gas stoves, hot water heating and without air conditioning when compared with those who had no

hobbies and lived in air conditioned homes with electric stoves and forced air heating. The effects in the other groups were inconsistent, and in adults the numbers in the sub-groups were too small for meaningful conclusions to be made.

4.3.5 Frequency of Observations Between Pairs of Exposure Variables

In general, homes with electric stoves had more forced air heating, hot water heating, electric heating and air conditioning. In addition, these homes had a greater proportion of smokers and residents with hobbies (Appendixes 4.3.1 to 4.3.4).

Crowded homes had a greater proportion of forced air heating, hot water heating and electric heating. These homes also had more air conditioning and a greater proportion of smokers. In air conditioned homes there was a smaller proportion of hot water and electric heating. Those residents who had hobbies lived in homes with fewer hot water and electric heating, less air conditioning, fewer smokers, and in addition they lived in homes that were crowded.

TABLES 4.16 - 4.20

MULTIFACTORIAL INTERACTIONS BETWEEN EXPOSURE VARIABLES

ON r FEV_1 AND SYMPTOMS IN CHILDREN AND ADULTS

LEGEND FOR FOLOWING TABLES (4.16 to 4.20).

a. Variables:

HB = Hobbies
HT = Heating
GAS = Gas Stoves
CK = Cooking Fuel
CR = Crowding

ELEC = Electric Stoves
No.S = Number of Smokers
AC = Air Conditioning
HW = Hot Water Heater
FA = Forced Air Heater

Zero'S = No Smoker in Home
1-S = One Smoker in Home
CR 1 = Homes With Less Than 5 Residents
CR 2 = Home With 5 Or More Residents

b. Subjects:

WM = White Males
WF = White Females

BM = Black Males
BM = Black Females

c. Level of Significance:

* = $P < .05$
** = $P < .01$
*** = $P < .001$

NS = Not Significant
\- = Not Sufficient data

d. CGH = cough
PGM = phlegm
WHZ = wheeze
DYS = dyspnea

TABLE 4.16

MULTIFACTORIAL INTERACTIONS BETWEEN EXPOSURE VARIABLES ON r.FEV$_1$ IN CHILDREN AND ADULTS

		CHILDREN	ADULTS
THREE VARIABLES:		ACxCK	
HB	WM	***	NS
	WF	***	*
	BM	**	--
	BF	**	NS
HT	WM	***	*
	WF	***	NS
	BM	***	--
	BF	***	**
CR	WM	***	NS
	WF	***	NS
	BM	***	--
	BF	***	**
NO.S	WM	***	NS
	WF	***	NS
	BM	**	--
	BF	**	NS
FOUR VARIABLES:		ACxCKxHTxHB	
	WM	***	*
	WF	***	NS
	BM	***	--
	BF	***	**

TABLE 4.17

MULTIFACTORIAL INTERACTIONS BETWEEN EXPOSURE VARIABLES ON SYMPTOMS IN CHILDREN AND ADULTS

		CHILDREN				ADULTS			
THREE VARIABLES		ACxCK	ACxCK						
		CGH	PGM	WHZ	DYS	CGH	PGM	WHZ	DYS
HB	WM	NS	NS	NS	NS	NS	NS	NS	*
	WF	NS	*	NS	***	NS	NS	NS	NS
	BM	NS	NS	NS	NS	--	--	--	--
	BF	***	***	NS	**	NS	NS	***	NS
HT	WM	NS	NS	NS	NS	NS	NS	NS	*
	WF	NS	NS	NS	NS	NS	NS	NS	NS
	BM	NS	*	NS	NS	--	--	--	--
	BF	NS	NS	NS	NS	***	**	NS	NS
CR	WM	NS	NS	NS	NS	NS	NS	NS	NS
	WF	NS	NS	NS	NS	NS	NS	NS	NS
	BM	***	NS	NS	NS	--	--	--	--
	BF	NS	NS	NS	NS	*	*	NS	NS
NO.S	WM	NS	NS	*	NS	NS	NS	NS	NS
	WF	NS	NS	*	NS	NS	NS	NS	NS
	BM	NS	NS	NS	NS	--	--	--	--
	BF	NS	NS	*	NS	**	*	NS	NS
FOUR VARIABLES:		ACxCKxHTxHB							
	WM	NS	NS	NS	NS	NS	NS	NS	NS
	WF	NS	***	NS	**	NS	NS	NS	NS
	BM	NS	NS	*	NS	--	--	--	--
	BF	***	***	NS	*	**	**	***	NS

TABLE 4.18

MEAN r FEV_1 FOR INTERACTION BETWEEN EXPOSURE VARIABLES IN CHILDREN (liters)

	WHITE				BLACK			
	MALE		FEMALE		MALE		FEMALE	
INTERACTIONS	NO.	MEAN	NO.	MEAN	NO.	MEAN	NO.	MEAN
NO.ACxGASxYesHB	51	.071	14	.044	12	.322	4	.392
YesACxELECxNoHB	171	.400	153	.430	56	.387	58	.287
No.ACxGASxHW	55	-.014	47	.006	5	.315	3	.399
YesACXELECxFA	136	.454	106	.483	27	.406	19	.370
No.ACxGASxI-S	64	.089	55	.064	73	.344	50	.365
YesACxELECxZero-S	78	.440	58	.400	35	.380	31	.225
No.ACxGASxCR 2	133	.034	106	.166	164	.360	161	.409
YesACxELECxCR 1	68	.431	62	.440	12	.366	17	.233
4-WAY								
NoACxGASxYesHBxHW	23	.040	10	.032	1	-.169	0	--
YesACxELECxNoHBxFA	25	.427	100	.493	20	.376	19	.370

TABLE 4.19

MEAN r FEV_1 FOR INTERACTION BETWEEN EXPOSURE VARIABLES IN ADULTS (liters)

	WHITE				BLACK			
	MALE		FEMALE		MALE		FEMALE	
INTERACTIONS	NO.	MEAN	NO.	MEAN	NO.	MEAN	NO.	MEAN
3-WAY								
NoACxGASxYesHB	12	.334	30	.064	--	--	2	--
YesACxELECxNoHB	77	.003	252	.004	--	--	40	.046
NoACxGASxHW	30	.167	90	.012	--	--	3	--
YesAcxELECxFA	54	.034	186	.014	--	--	13	.136
NoACxGASx1-S	18	-.116	86	.029	--	--	1	--
YesACxELECxZero-S	63	-.021	166	-.019	--	--	5	--
NoACxGASxCR 2	30	.065	75	-.031	--	--	77	.189
YesACxELECxCR 1	63	-.018	219	-.014	--	--	16	.037
4-WAY								
NoACxGASxYesHBxHW	7	.404	16	.042	--	--	0	--
YesACxELECxNoHBxFA	48	.042	167	.021	--	--	13	.136

TABLE 4.20

DIFFERENCES BETWEEN INTERACTION GROUPS FOR

FEV_1 (IN LITERS) IN WHITE CHILDREN

	MEAN DIFFERENCES IN FEV_1 FROM UNIVARIATE TABLES	
EXPOSURE VARIABLES	MALES	FEMALES
NoHB-YESHB	.06	.12
YesAC-NoAC	.21	.25
ELEC-GAS	.17	.08
FA-HW	.27	.27
ZeroS- 1-S	.01	.03
CR 1 - CR 2	.12	.05

	MEAN FEV_1 BY INTERACTION		MEAN FEV_1 BY ADDITION +	
3-WAY	MALES	FEMALES	MALES	FEMALES
(YesACxELECxNoHB)-(NoACxGASxYesxHB)	.329	.386	.44	.45
(YesACxELECxFA)-(NoACxGASxHW)	.468	.489	.65	.60
(YesACxELECxZeroS)-(NoACxGASx1-S)	.351	.336	.39	.36
(YesACxELECxCR 1)-NoACxGASxCR 2)	.397	.274	.50	.38
4-WAY				
(YesACxELECxNoHBxFA)- (NoACxGASxYesHBxHW)	.387	.461	.71	.72

+ obtained by adding the individual means from above table.

CHAPTER 5 - RESULTS II

DOMESTIC AIR POLLUTION EXPOSURE IN A GROUP OF HOUSEWIVES

5.1 Sampler Validation Data

5.1.1 Sampling Schedule

In order to define the domestic air pollution exposure, a sampling period of 24 hours was undertaken once in the summer and once in the winter for each matched pair of subjects. I hoped that this schedule would allow at least some initial insights into the possible magnitude of domestic air pollutants. To investigate the feasibility of using 24-hour samples to define the domestic environment, two direct readings (i.e. for NO_2 and TSP) were tested in the same environments for up to six consecutive days. The data on Table 5.1 indicated (by using one way analysis of variance) that there were no differences between days for TSP in two homes without smokers, whereas there was a significant difference in NO_2 between days in a home with a gas stove, and a just significant difference in a home with an electric stove.

5.1.2 Intersampler Correlation

Two personal environment samplers (PES I and PES II) were initially constructed and the collection characteristics of these were compared against each other and with an outdoor environment sampler (OES).

The results for SO_2 and NO_2 (Appendices 5.1 and 5.2) showed very good correlation between samplers i.e. r = 0.99 and 0.96 respectively. For respirable suspended particulate (RSP), some dis-similarities between samplers were found especially when particulate levels fell below 30 ug/m^3. The correlations were still high though (r = 0.78). (Appendix 5.3).

5.1.3. Selection of Filter Medium for Particulates.

The medium most widely used for collecting airborne particulate matter is cellulose nitrate membrane. In the preliminary tests it was found that this particulate data was very variable because of the normal fluctuation in humidity and the high static charge which is a characteristic of membrane filters. A number of cellulose nitrate (CN) filters were weighed at 3 intervals 24 hours apart; the variability in the weights was found to be high. Other researchers found the same and blamed the hydroscopicity and the high static charge for the discrepancies (Mark et al, 1974; Tierney et al, 1967; and Cahn, 1963). Polyvinyl chloride membrane filters are less hydroscopic but because of the high static charge, the results of these were also found to be variable. The use of glass filter (GF) filters was then investigated and these were found to give reproducible results. In the tests comparing GF, CN and PVC filters that were dessicated for 24 hours at 45°C and weighed, the GF showed little fluctuation, but wide variations (up to 100 ug) occurred with the membrane filters (Appendix 5.4).

5.1.4 Summary

i. In the tests undertaken to determine whether the 24-hour sampling period would be representative of most days in the domestic environment, it was found that the particulate levels in homes with gas and electric stoves and with no smokers showed no differences in levels between days, whereas the NO_2 levels showed significant variations between days in non-smokers' homes with gas and electric stoves. The significant daily variation in NO_2 was due to single days of high levels; when these days were removed and the analyses repeated, no variation between days were observed.

ii. Comparisons between two personal samplers and one outdoor sampler stationed in the same environment and with sampling probes at the same height above ground level showed very good correlation. This was as expected because of the similarities in collecting media, the differences being mainly in the types of sampling pumps used e.g. the PES were battery-operated while the OES was AC-operated. The flow rates for SO_2 and NO_2 were almost the same, but for RSP, there were some differences e.g. PES was 1.7 lpm while OES was 900 lpm. For RSP, both samplers had the same collection characteristics and size cut-off.

iii. Glass fiber filters were chosen as the collection media for respirable particulates because of the high collection efficiency, independence of electrical charge and relative humidity, and their usefulness for further chemical analyses.

Table 5.1

Daily Variation of TSP and NO_2 in Domestic Environments.

Mean 24-hour

Concentration	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	F-test*	SIGNIFICANCE
TSP+								
Non-Smoker	.027	.024	.015	.036	.021	-	1.53	NS
Electric								
$NO_2(ug/m^3)$								
Non-Smoker	38	36	30	26	44	-	2.59	<.05
Electric								
TSP+								
Non-Smoker	.062	.053	.033	.029	.067	.065	0.50	NS
gas								
$NO_2(ug/m^3)$								
Non-Smoker	456	288	480	714	398	-	11.33	<.01
Gas								

* F-value from one way analysis of variance.

\+ TSP are expressed in COH Units (coefficient of haze). For this study, a continuous particulate tape sampler was used.

5.2 DOMESTIC AIR POLLUTION DATA

5.2.1 Indoor - Outdoor Comparisons

The air pollution summary data for SO_2, NO_2 and RSP are given as geometric means since it has been found that air pollution levels usually follow a log-normal distribution.

The summary air pollution exposure data for the housewives in Lebanon and Winnsboro are shown in tables 5.2a and 5.2b. When the data for the housewives with symptoms were compared against those without symptoms, no statistically significant differences were found (Table 5.3). To determine whether the domestic exposure was different from the outdoor exposure data, for both housewives groups were combined and compared against the outside air pollution data; this comparison revealed in general that the SO_2 levels were usually higher outdoors, whereas the NO_2 and RSP levels were higher indoors (Table 5.3).

In Lebanon, the homes of smokers showed higher levels of NO_2 and RSP than the homes of non-smokers. In general the winter air pollution levels in the Lebanon homes were higher than the summer levels. In Winnsboro, the homes of smokers did not show any consistent trends, nor were there any trends between the levels in the homes during winter or summer.

TABLE 5.2a

COMPARISON OF DOMESTIC AND OUTSIDE ENVIRONMENTS FOR AIR POLLUTANTS, LEBANNON, CONNECTICUT

Pollutant	Smoking Category (# of pairs)	Season	Exposures (ug/m^3)* Domestic: Airway Disease	Domestic: Control	Domestic: Disease and Control Combined	Outside
SO_2	non-smokers (7)	Winter	6.2 ± 1.7	4.6 ± 1.9	5.3 ± 1.9	17.6 ± 2.0
		Summer	4.2 ± 2.2	1.6 ± 6.8	2.6 ± 4.5	8.7 ± 1.5
	smokers (7)	Winter	1.9 ± 7.6	3.5 ± 7.8	1.0 ± 8.9	6.2 ± 1.8
		Summer	0.6 ± 10.3	1.7 ± 8.1	1.0 ± 8.9	1.6 ± 3.7
NO_2	non-smokers (7)	Winter	80.9 ± 1.5	74.2 ± 1.6	77.5 ± 1.9	63.9 ± 1.3
		Summer	86.4 ± 1.8	88.6 ± 1.5	87.5 ± 1.6	51.7 ± 1.4
	smokers (7	Winter	206.7 ± 1.9	128.6 ± 1.9	163.0 ± 1.9	43.0 ± 1.7
		Summer	103.9 ± 1.4	124.1 ± 2.1	113.6 ± 1.8	16.1 ± 1.7
RSP	non-smokers (7)	Winter	54.2 ± 3.1	38.6 ± 2.0	45.8 ± 2.5	27.8 ± 1.5
		Summer	39.6 ± 1.5	31.0 ± 1.6	35.1 ± 1.5	31.5 ± 1.5
	smokers (7)	Winter	101.6 ± 1.7	105.3 ± 1.9	103.4 ± 1.8	22.6 ± 1.6
		Summer	103.8 ± 1.4	91.2 ± 1.5	97.3 ± 1.4	28.7 ± 1.9

* in geometric mean ± geometric standard deviation.

TABLE 5.2b

COMPARISON OF DOMESTIC AND OUTSIDE ENVIRONMENTS FOR AIR POLLUTANTS, WINNSBORO, SOUTH CAROLINA

Pollutant	Smoking Category (# of pairs)	Season	Exposures (ug/m^3)#			
			Domestic			Outside
			Airway Disease	Control	Disease and Control Combined	
SO_2	non-smokers (4)	Winter	5.3 ± 5.0	9.6 ± 5.0	5.3 ± 4.4	23.3 ± 2.2
		Summer	1.3 ± 1.7	ND	1.1 ± 1.5	1.7 ± 2.8
	smokers (8)	Winter	2.8 ± 2.6	4.9 ± 3.5	3.7 ± 3.1	36.6 ± 1.6
		Summer	ND	ND	ND	1.5 ± 2.3
NO_2	non-smokers (4)	Winter	70.9 ± 1.6	75.9 ± 1.3	73.3 ± 1.4	17.9 ± 1.1
		Summer	48.6 ± 3.5	86.9 ± 2.7	65.0 ± 3.0	18.3 ± 1.2
	smokers (8)	Winter	95.7 ± 2.1	103.1 ± 1.9	92.5 ± 1.7	17.6 ± 1.4
		Summer	87.1 ± 1.4	82.9 ± 1.7	91.3 ± 1.8	21.1 ± 1.3
RSP	non-smokers (4)	Winter	58.0 ± 1.1	50.6 ± 2.6	35.6 ± 3.1	31.1 ± 75.4
		Summer	33.1 ± 3.1	25.0 ±31.6	59.5 ± 1.4	75.4 ± 1.3
	smokers (8)	Winter	54.1 ± 1.6	42.3 ± 2.7	45.0 ± 3.3	21.8 ± 1.9
		Summer	78.5 ± 1.4	48.0 ± 4.4	65.2 ± 1.5	55.5 ± 1.4

in geometric mean ± geometric standard deviation. ND = Not detected

TABLE 5.3

AIR POLLUTION DIFFERENCES BETWEEN HOMES WITH NORMAL AND SYMPTOMATIC HOUSEWIVES

			Normal vs. Disease			Disease & Normal vs. Outside		
			SO_2	NO_2	RSP	SO_2	NO_2	RSP
LEBANON	Non-Smokers	Winter	-	-	-	***	***	***
		Summer	-	-	-	**	***	***
	Smokers	Winter	-	-	-	x	***	***
		Summer	-	-	-	-	***	***
WINNSBORO	Non-Smokers	Winter	-	-	-	***	***	***
		Summer	-	-	-	-	***	**
	Smokers	Winter	-	-	-	**	***	***
		Summer	-	-	-	-	***	***

Levels of Significance:

- = not significant
x = $P < .05$
** = $P < .01$
*** = $P < .001$

5.2.2 Sources of Nitrogen Dioxide

Twenty one percent of the homes had gas stoves, and in these homes the nitrogen dioxide levels (Table 5.4) were significantly higher than homes with electric stoves ($P<.001$). This differential between homes with gas and electric stoves was seen in both seasons. The winter NO_2 levels were higher than the summer in homes with both gas and electric stoves. Homes with gas stoves and air conditioning had less NO_2 than homes without air conditioning, but the numbers in each group were too small for meaningful comparisons (Table 5.5).

Five percent of the homes had gas radiant heaters, but some of these homes also had gas stoves, hence it was not possible to assess the emissions of NO_2 from this source. The homes of smokers without gas stoves had more NO_2 than homes of non-smokers without gas stoves. Although the differences were significant ($P< .001$ and $P< .02$ respectively), the absolute differences in the geometric means were small (Table 5.6).

The tests conducted to determine the daily variations in domestic NO_2 loading showed that in two homes with gas ranges, the NO_2 levels were sometimes in excess of 1000 ug/m^3 (i.e. 2-hourly values). In one home, thirteen of the sixty 2-hourly values were in excess of 1000 ug/m^3 and in the other home, eight of the sixty 2-hourly values were in excess of 1000 ug/m^3. In this latter home, peak 2-hourly values in excess of 3000 ug/m^3 were observed on two separate occasions. In all cases, these peaks were directly related to extensive use of the gas stoves and ovens.

TABLE 5.4

Nitrogen Dioxide Levels in Homes+ with Gas and Electric Stoves

	Gas		Electric	
	Winter	Summer	Winter	Summer
No. of obs	11	11	41	41
Geometric Mean (ug/m^3)	214.7	179.5	80.6	75.6
Geometric Standard Deviation	1.97	1.56	1.62	1.78
Unpaired t-test		$P<.001$		

+ These levels include homes in Lebanon and Winsboro.

TABLE 5.5

Nitrogen Dioxide Levels in Homes with Gas and Electric Stoves with and without Air Conditioning

(Summer Levels Only)

	With Air Conditioning		Without Air Conditioning	
	Electric	Gas	Electric	Gas
No. of obs.	12	4	29	7
Geometric Mean (ug/m^3)	70.9	175.1	81.8	182.1
Geometric Standard Deviation	2.00	1.56	1.70	1.62

TABLE 5.6

Nitrogen Dioxide Levels in Homes without Gas Stoves as a Function of Smoking

	Winter		Summer	
	Smoker	No Smoker	Smoker	No Smoker
No. of obs.	29	12	29	12
Geometric Mean (ug/m^3)	82.6	75.7	76.1	74.5
Geometric Standard Deviation	1.75	1.23	1.74	1.93
Unpaired t-test	$P < .001$		$P < .02$	

5.2.3 Sources of Particulates

Cigarette smoking contributed significantly to the RSP loading within homes in the winter and summer (Table 5.7). The presence of one smoker in the home resulted in significantly more RSP than homes with no smokers ($P<.001$), and homes with 2 or more smokers showed more RSP than homes with one smoker or no smokers ($P<.001$). In homes with air conditioning (Table 5.8), the RSP levels were higher in both homes with zero smokers ($P<.01$) and with at least one smoker ($P<.001$) compared with homes without air conditioning.

Carpeted houses with at least one smoker had more RSP than non-carpeted houses with at least one smoker (Table 5.9), but houses of non-smokers with carpet had less RSP than houses of non-smokers without carpets.

Homes with hot water heating with no smokers had significantly higher levels of RSP (geometric mean 66.7ug/m^3) than homes heated by forced air (geometric mean 37.7ug/m^3). Homes with one or more smokers and with hot water heaters had significantly higher levels of RSP (geometric mean 84.8ug/m^3) than homes heated by forced air (geometric mean 57.1ug/m^3).

TABLE 5.7

Respirable Suspended Particulates as a Function of Smoking

	Number of Smokers in Home					
	Winter			Summer		
	0	1	⩾2	0	1	⩾2
No. of obs.	14	18	18	14	18	18
Geometric Mean (ug/m^3)	40.9	47.9	75.6	35.2	56.8	84.7
Geometric Standard Deviation	2.83	2.47	2.96	1.67	1.79	1.50
Unpaired t-test		P<.001	P<.001		P<.001	P<.001

TABLE 5.8

Respirable Suspended Particulates Exposure of Smokers and Non Smokers as a Function of Air Conditioning (AC)

(Summer Levels Only)

	Zero Smokers in Home		At least one Smoker in Home	
	AC	No AC	AC	No AC
No. of obs	4	11	11	25
Geometric Mean (ug/m^3)	34.3	32.5	80.5	70.11
Geometric Standard Deviation	1.60	1.84	1.40	1.64
Unpaired t-test	$P < .01$		$P < .001$	

TABLE 5.9

Respirable Suspended Particulates Exposure of Smokers in Houses with and without Carpets

	Smoker Home		Non Smoker Home	
	Carpeted	Non-Carpeted	Carpeted	Non Carpeted
No. obs.	28	8	9	7
Geometric Mean (ug/m^3)	76.6	70.2	38.7	53.7
Geometric Standard Deviation	1.68	2.14	1,91	1.73
Unpaired t-test	$P<.001$		$P<.001$	

5.2.4 Summary

The use of gas stoves accounted for most of the NO_2 found within the homes. Cigarette smoking accounted for most of the RSP levels indoors. NO_2 and RSP levels were higher indoors whereas SO_2 was higher outdoors. Homes heated by hot water heating had higher levels of RSP than forced air heated homes.

5.3 Influence of Indoor Exposure Factors on Health

A total of 26 subjects were found who fitted the selection criteria i.e. housewives who had respiratory symptoms. These were matched with 26 housewives who had no symptoms.

5.3.1. Use of Gas Stoves

To determine whether the NO_2 exposure from gas stoves resulted in impairment of lung function, non-smokers in homes with gas stoves were compared with their counterparts in homes with electric stoves (Table 5.10). Housewives using gas stoves had significantly lower $MF_{25\%}$, $MF_{50\%}$, and FEV_1, but the number of housewives in the gas stove user group was small (N=3).

In the housewives study group, 31% of the subjects with symptoms used gas stoves, whereas 12% who used electric stoves showed symptoms. Comparing all subjects within these homes, then 28% of the gas stove users showed symptoms whereas 18% of electric stove users showed symptoms. Both these comparisons included smokers and non-smokers.

Table 5.10

Effect of Gas Stove Cooking on the Lung Function of Non-Smoking Housewives

Mean Lung Function + Standard Deviation #

	N	$MF_{25\%}$	$MF_{50\%}$	FEV_1
Gas-Stoves	3	-32.5±25.0**	-32.0±26.0**	-25.3±26.2*
Electric Stoves	19	- 1.3±15.9	- 2.7±13.7	- 2.2±14.9

Un-paired t-test between gas and electric stove users: * = $P < .05$; ** = $P < .01$

+ the mean values reported are the percent deviation from predicted.

5.3.2 Smoking

The lung function of non-smokers who lived in homes with (a) no smokers, (b) a home where one pack per day was smoked, and (c) a home where two or more packs per day were smoked, were compared. In this comparison the groups were separated into those who lived in homes with electric stoves and those who lived in homes with gas stoves. In general, the lung function of non-smokers whose exposure to NO_2 was low was not influenced by passive smoke (Table 5.11).

The lung function of non-smokers exposed to NO_2 from gas stoves did not appear to be affected by passive smoke but the numbers in each smoking category was small (Table 5.11). The lung function of non-smokers who were exposed both to NO_2 and passive smoke showed a greater deviation from predicted than non-smokers exposed to passive smoke alone, but the differences between these groups were not statistically significant.

An attempt was made to determine whether non-smoking subjects living with non-smoking or smoking symptomatic subjects had different lung function from non-smoking subjects in homes of normal subjects. Table 5.12 shows that there were no differences in lung function between non-smoking subjects living in homes with either symptomatic or normal subjects.

TABLE 5.11

LUNG FUNCTION OF NON-SMOKERS LIVING IN HOMES OF SMOKERS AND NON-SMOKERS AS A FUNCTION OF COOKING FUEL USED

Pks/day # Electric Stove Home	N	Mean ± Standard Deviation + MF 25%	MF 50%	FEV_1
0	36	- 4.0±15.7	- 0.2±12.4	- 1.9±13.3
1	27	- 6.7±16.5	- 3.0±12.0	-0.6±15.8
2 +	23	- 5.9±16.0	- 1.6±10.9	1.4±23.9
Pks/day # Gas Stove Home	**N**	**MF 25%**	**MF 50%**	**FEV_1**
0	6	-12.6±21.2	- 8.5±25.3	- 9.0±20.9
1	2	-27.2±9.1	-14.6±6.1	- 9.8±11.3
2 +	11	- 7.0±14.8	- 7.9±11.1	- 6.3±14.1

\# Packs of cigarette smoked indoors.

0 = no smoker in the home.

1 = 1 pk/day of cigarette smoked in the home.

2+ = 2 or more pks/day of cigarette smoked in the home.

\+ = See footnote, Table 5.10

There were no significant differences between the smoking groups for each lung function variable.

TABLE 5.12

LUNG FUNCTION OF SUBJECTS LIVING IN HOMES WITH SYMPTOMATIC AND NORMAL SUBJECTS (ALL NON-SMOKING)+

		Mean Lung Function ± Standard Deviation +		
Non-Smoking Housewives Group	N	$MF_{25\%}$	$MF_{50\%}$	FEV_1
Subjects in Home with Symptomatic Mother	15	- 4.9±13.9	- 3.2±15.5	- 5.1±15.8
Subjects in Home with Normal Mother	19	- 6.1±15.9	- 0.1±11.4	- 1.9±16.4
Smoking Housewives Group				
Subjects in Home with Symptomatic Mother	20	- 4.6±18.2	- 2.9±10.9	- 5.6±14.9
Subjects in Home with Normal Mother	23	- 8.1±15.3	- 4.5±9.4	- 1.7±21.8

+ The mean values shown in this table are for non-smokers only. It excludes the normal and symptomatic housewives lung function data.

No differences between subjects living in home with normal or symptomatic housewives.

+ See footnotes, Table 5.10.

5.3.3 Summary

The lung function of non-smokers was not influenced by passive smoking, nor by a combination of passive smoking and NO_2 from gas stoves. Non-smokers in the housewives group who had gas stoves had worse lung function than their counterparts with electric stoves. The probability that a significant difference would emerge from this analysis was low because of the small number of subjects in each group.

5.4 Correlation of Domestic Air Pollution with Lung Function

5.4.1 Simple Regression

The degree of correlation between the indoor air pollutants and lung function were investigated for (a) the housewives group (smokers and non-smokers) and (b) for all non-smoking residents. The analyses were conducted for NO_2 and RSP and the lung function parameters of $MF_{25\%}$, $MF_{50\%}$ and FEV_1. SO_2 was not compared because of the extremely low indoor levels. The correlation was conducted between the log of the pollution values and the percent deviation of the lung function measures from the predicted values.

For non-smoking housewives, there were negative correlations between log NO_2 and the lung function parameter, but none of these were statistically significant. (Table 5.13) This group also showed negative correlations between RSP and the lung function parameters, two of them being statistically significant i.e. $MF_{25\%}$ ($P < .05$) and $MF_{50\%}$ ($P < .05$) (Table 5.13). When all residents of the homes were studied, the effect of NO_2 on lung function was negligible, but the effect of RSP was significant for $MF_{50\%}$ ($P < .03$).

TABLE 5.13

CORRELATION BETWEEN INDOOR POLLUTANTS AND LUNG FUNCTION OF NON-SMOKERS AND SMOKERS

	Correlation Coefficient (r)					
Non-Smokers	Housewives only			All residents in the home		
	$MF_{25\%}$	$MF_{50\%}$	FEV_1	$MF_{25\%}$	$MF_{50\%}$	FEV_1
NO_2	- .29	- .34	- .23	- .07	- .04	- .04
RSP	- .41*	- .41*	- .31	- .28	- .45*	- .39*
Smokers						
NO_2	.14	.25	- 13	.09	.14	- .27
RSP	- .09	- .19	- .11	- .06	- .30	- .31

+ The correlation was done on the log of the pollution concentration and the percent deviation of the lung function value from the predicted value.

* $P < .05$

For smokers there were no associations between exposure to NO_2 and RSP and lung function; an indication that the effect of smoking may overide any effects of the other pollutants in the home (Table 5.13). There was no association between the NO_2 and RSP values ($P<.05$).

5.4.2 Multiple Regression

An attempt was made to determine whether the pollutants, NO_2 and RSP, acting together would explain more of the effects on the lung function parameters. A SAS (Statistical Analysis System) data set was created from the log values for NO_2 and RSP and for the percent deviation of the lung function parameters, $MF_{25\%}$, $MF_{50\%}$ and FEV_1 from their predicted values. Multiple regression analyses were conducted on smokers and non smokers, and on housewives and all residents of the homes.

The regression analysis for non-smoking housewives showed that RSP explained a larger proportion of the variation than NO_2 on all lung function measurements (Table 5.14.) When both NO_2 and RSP were included in the multiple regression, it was found that a larger proportion of the variation was explained than when both pollutants were analysed separately. It was also found that RSP was a better predictor of $MF_{25\%}$ ($P<.03$) and $MF_{50\%}$ ($P<.02$), and that RSP was acting separately to explain the effects on the lung function measurements.

When all the non-smoking residents were tested, RSP was again found to have the major effect on lung function, especially on $MF_{50\%}$; the effect of NO_2 was less predominant than in the housewives group (Table 5.14)

For Smoking housewives, and for all smoking residents, the multiple regression analysis did not show any significant effects between lung function and the pollution values.

Multifactorial analyses of variance were conducted on the NO_2 and RSP levels and the lung function values to determine if there were any significant interactions; the analyses revealed no interaction between the pollutants.

5.4.3. Summary

Simple linear regression analysis between indoor pollutants and lung function in non-smokers showed that RSP correlated well with lung function depression. The multiple regression analysis indicated that both pollutants acted separately on the lung, but RSP was a better predictor of lung function than NO_2.

TABLE 5.14

Multiple Regression of Lung Function on NO_2 and RSP of Non-Smokers and Smokers

Variable	Percent of Variation Explained by Variable (R^2) HouseWives Only			All Residents		
	$MF_{25\%}$	$MF_{50\%}$	FEV_1	$MF_{25\%}$	$MF_{50\%}$	FEV_1
Non-Smokers						
NO_2	8	11	5	1	1	6
RSP	17*	17*	9	8	20*	15
NO_2 x RSP	29*[1]	34*[1]	17	9	20*[1]	15
Smokers						
NO_2	2	6	2	1	2	7
RSP	8	4	1	1	9	9
NO_2 x RSP	3	11	3	1	12	15

* $P < .05$

1 Only RSP was acting separately to explain the lung function.

CHAPTER 6

DISCUSSION

The results from this study have shown that the nine domestic factors chosen were associated to a varying extent, singly and in combination, with lung function depression and excess reporting of symptoms. Actual air pollution measurements in the domestic environment have shown it to be an independent source of respiratory irritants, and in certain cases, these measurements have supported the associations between the domestic factors and the dependent variables. These findings will be discussed in terms of their implications in the overall epidemiology of respiratory diseases, and in terms of what other researchers have found in related studies.

6.1 Domestic Factors

The keeping of pets in the home could result in exposure to airborne dusts which may cause respiratory irritation. From this study, this source of exposure did not consistently show any effects on respiratory health of any of the population subgroups. Exposure to particles from pets could result in atopic respiratory responses (Korsgaard, 1982), but there was no increased reporting of wheeze in this study group. It is possible that people with sensitivity to pets may be forced to get rid of them and thence the reporting in the study might be unbalanced. It was not possible to determine whether subjects did have to give up pets for health reasons, but the mean lung function values of both the exposure groups for children and adults were similar in most regards.

When the pet variable was included in the multiple regression, the lack of consistent significant trends indicated that this variable was not an important predictor, either by itself or in combination in the causation of chronic respiratory disease in children and adults.

The fireplace variable was included in this study since it was thought that leakage of gases, vapours and dusts from the fireplace would have resulted in chronic exposure with the resultant effect on respiratory health. But the findings from the study indicated that users of fireplaces have in general similar lung function and symptom reporting frequency rates as those who did not have fireplaces. Even when this variable was considered with the others in the multiple regression analyses, it was not found to be an important predictor of chronic respiratory diseases. In some subgroups, the use of fireplaces was associated with beneficial effects which may be due to the fact that the use of open hearth fireplaces may result in better ventilation of the home with the chimney acting as an exhaust system for other airborne gases, vapours and dusts from the home. In a follow-up study supported by the World Health Organization in Toronto, it was found that homes with fireplaces had lower particulate concentrations than homes without fireplaces (Silverman et al, 1982).

Damp humid homes have been shown to harbour more mites (Korsgaard, 1982) and to cause more respiratory diseases (Korsgaard, 1982, Holma, 1980). These two studies were conducted in Europe; but I was unable to find in the literature similar reporting in North American studies. In fact, in North American homes, humidification is deliberately added during the

winter time, and although this might be a potential source of viable aerosols, the findings in this study did not show any significant excess symptoms or depressed lung function in those who reported humidifier usage. Humidifiers may also be added for therapeutic reasons for those suffering from chronic wheezing. The findings for children and adults indicate a slight excess (though not statistically significant) of wheeze reporting by those whose houses had humidifiers. When this variable was included in the multiple regression model, it was found that, in general the use of humidifiers had positive effects on lung function, and reduced symptom reporting. The use of humidifiers may have also a beneficial effect on the lung by providing the necessary humidification for efficient functioning of the defense mechanisms. In the World Health Organization Study in Toronto, 12 of 13 asthmatics had humidifiers whereas 10 of 13 controls had humidifiers (Silverman et al, 1982).

Although hobbies at home can cause exposure to airborne gases, vapours and dusts which may cause irritation of the respiratory tract, there was no evidence in the literature that this factor was investigated before. From this study, hobbies were found to be important making a separate contribution to the depression of lung function and increased reporting of symptoms. The depression in FEV_1 was small, but seemed to be consistent in the multiple regression analyses and in the multifactorial analyses of variance. It showed significant two-way interaction with most of the other variables, three-way interaction with cooking fuel and air conditioning, and four-way interaction with cooking fuel, heating fuel and air conditioning.

The use of air conditioners may be beneficial in respiratory health because of the reduced diffusion of outside pollution to the indoors (Dockery et al, 1981). This was reflected in the groups of children in this study whereby the mean FEV_1 of white boys and girls and black boys who lived in air conditioned homes was significantly better than their peers who lived in non-air conditioned homes. This exposure variable was found to act separately when the other exposure variables were considered in the multiple regression analysis on the FEV_1 of the children.

The multifactorial analysis showed that white children who lived in houses without air conditioning, but with other potential sources of air pollution, had lower FEV_1 than those who lived in homes with air conditioning and without the potential sources such as hobbies, gas cooking, hot water heating, crowding and smokers. The fact that the same effects were not seen in adults may be because adults have had other exposures that would have affected respiratory health and that they may have acquired the air conditioner at a later point in their life. The levels for NO_2 in the air conditioned homes in the case-control part of this study showed that homes with gas and electric stoves had slightly lower levels than non-air conditioned homes probably because of lower diffusion from outside and more mixing of air within the air conditioned homes. In the case of homes with and without smokers, the air conditioned homes had slightly higher levels of particulate matter than the non air conditioned homes indicating an accumulation of suspended particulate matter within air conditioned homes; the differences in the means were too small though to be of any

clinical significance. In the WHO study in Toronto, we found that air conditioned homes had lower particulate levels than non-air conditioned homes (Silverman et al, 1982).

Homes with hot water heating had higher levels of RSP probably because of the thermal convective currents which caused resuspension of particulate matter. It is also possible that these homes were older and therefore more prone to leaks; older homes may have more dust generated by the aging of the building materials. Hot water heated homes may also be drier which may encourage breakdown of materials. The dustier environment may account for the consistent findings of lower FEV_1 and more symptoms in most of the subject groups. It is also possible that the drier air in these homes may cause respiratory tract irritation (Anderson et al, 1973) which may be aggravated in the presence of airborne gases and dusts. This was evident in the multifactorial analyses, in that, when the heating variable was considered with the other variables that may affect respirtory health, there was an amplified effect. Yarnell et al (1979), in their study in Wales, found that central heating, which was interpreted to mean hot water heating, was associated with lower FEV_1 in children when compared to those who lived in homes with coal fuel heating. It was interesting to note that there were fewer asthmatic children in the centrally heated homes which may support the theory that these homes were dry thus making it less likely that residents would be exposed to molds and mites. Lave and Seskin (1972) found that hot water heating was associated with increased mortality in the United States.

In this study, homes with gas stoves had significantly more NO_2 than homes with electric stoves. In some of these homes the peak levels reached as high as 3000 ug/m^3 when there was extensive use of the gas stoves and ovens. In all the studies to date (Keller et al, 1979; Melia et al, 1979; Goldstein et al, 1979), the NO_2 levels in homes with gas stoves were higher than homes with electric stoves. In this study, the levels of NO_2 in the gas stove home were approximately 100 ug/m_3 more than in homes with electric stoves.

The effect of NO_2 on lung function was only evident in white children. In adults, it is possible that exposure to other airborne irritants may overshadow the effect of exposure to NO_2 from gas stoves. In black children, there were no effects from the potential exposure to NO_2 from gas cooking probably because black children may spend less time in the vicinity of the kitchen. I did not collect sufficient air samples in the homes of black residents to determine whether the NO_2 trends were any different when compared to the homes of white residents. The other researchers who measured NO_2 in homes did not indicate whether any of the measurements were conducted in the homes of black residents; and in the health effects studies in the literature, no black populations seem to have been studied.

The effects on children shown in this study were also shown by Melia et al (1977 and 1979), Hasselblad et al (1981), Dockery et al (1982), and Dodge (1982). Dockery et al found the mean decrease in FEV_1 of children in gas stove homes to be less than 20 mls whereas in my study the decline in white boys was 170 mls, and in girls it was 80 mls. The

negative results found by Keller et al (1979a) might be due to the fact that only acute respiratory illness was assessed rather than chronic respiratory disease symptoms.

The potential exposure to NO_2 from gas stoves resulted in further decreases in FEV_1 when other exposure factors were added, indicating that the pollutants were acting together to cause an aggravated reaction in the respiratory tract. The interaction was strongest with the heating and air conditioning variables, and weakest with the number of smokers variable. In the case-control study, the interaction between NO_2 and RSP was absent. Both pollutants acted separately in explaining the changes in lung function with the RSP explaining more of the changes in lung function than NO_2.

Domestic crowding has been associated with increased reporting of respiratory symptoms (Reid et al, 1958; Holland et al, 1980; Colley et al 1974; Leeder et al 1977; and Davis et al 1980). It is possible that crowded homes may have more activity thus resulting in the generation of more airborne gases and dusts. Crowded homes had more smokers than non-crowded homes, but the interaction was weak on symptoms and lung function. Residents of crowded homes may transmit infection which may result eventually in increased respiratory diseases (Coffin et al, 1968; Fletcher et al, 1977; Lebowitz et al, 1978; Yarnel et al, 1981). Monto and Ross (1978) found that bronchitic subjects had more respiratory infections which may spread to others. It is possible that there might be lung function resemblance between siblings in the same home thus accounting for some of the findings in this study. But when the

correlation of residual lung function between brothers and between sisters were undertaken, the only significant correlation was between sisters for r FEV_1 ($r=.26$, $P<.05$) (Schilling et al, 1977).

In this study it was not possible to compare the air pollution levels in homes of the various crowding groups because of the small numbers in each sub-category. In the univariate analyses, the effect of crowding was evident in white children but only to a small extent when compared to the other exposure variables. In black children, crowding had no effects whatsoever and I am unable to give a logical explanation for this finding. In adults it is possible that the effects of other exposures to airborne gases and dust would have removed the differences between the groups. In white children, the interaction between crowding and the other exposure variables resulted in additional reductions in FEV_1.

The presence of one smoker in the home increased the RSP loading by 8 ug/m^3 in the winter and 20 ug/m^3 in the summer; two smokers in the home increased the levels by 35 and 50 ug/m^3 respectively. Dockery et al (1981) found similar increases in the Harvard six-city study. Repace (1980) and Weber (1980) found levels very much in excess of these i.e. 600 ug/m^3 and 100 ug/m^3 respectively. Smoking in air conditioned homes resulted in slightly higher levels of RSP compared to homes without air conditioning probably because of the lack of outside air dilution. Carpeted homes with smokers had higher levels of RSP than non-carpeted homes because smoke particles may accumulate in the carpet and become resuspended during activities in the home. Cigarette smoking

indoors contributed about 2 ug/m^3 of NO_2 in the summer and 7 ug/m^3 in the winter. This NO_2 may come from the oxidation of atomospheric nitrogen at the glowing point in the cigarette.

The evidence for respiratory health effects from passive smoking is somewhat controversial. Some researchers have implied that smoking subjects contract respiratory diseases which may be transferred to other residents in the home thus accounting for the effects of passive smoking (Colley et al, 1974; Lancet editorial, 1974; Monto et al, 1975; Leeder et al, 1976; Tager et al, 1976). Other researchers found no associations between passive smoking and respiratory diseases (Tager et al, 1976; Yarnel and St. Leger, 1981; Binder et al, 1976). In a follow-up of the Tager et al (1976) study it was found that passive smoking did have an effect on a lung function score which was able to detect very small changes in lung function. Other workers found passive smoking to be a cause of respiratory diseases (Weiss et al, 1980; Hasselblad et al, 1981; Lebowitz et al; Harlap, 1974; Ferguson et al, 1980; Shenker et al, 1981; Cameron et al, 1969; Cameron and Robertson, 1973; Comstock et al, 1981; White and Froeb, 1980; Dockery et al, 1982; Dodge et al, 1982; Kauffmann, 1980; Kauffmann et al 1983). The evidence on passive smoking was shown for only some sub-groups e.g. young children, and in some cases, only in one sex. In my study, the effect of passive smoking on lung function and symptoms was weak in children and adults with the main effects being seen primarily with one smoker but not with two smokers in the home. The air sampling data showed that the contribution of smoking within the home was a real contributor to RSP, but it is possible that these levels are not high enough to be of

clinical significance. In this study, it was not possible to compute the number of cigarettes smoked within each home; this would have been a better measure of exposure. It was also not also possible to determine whether the smoker in the home was the mother or father. In the studies which showed an effect of smoking, the association with maternal smoking was stronger.

The effect of passive smoking on lung cancer (Hirayama, 1981; Trichopoulos, 1981) was convincing, but the carcinogenic agents might have been low levels of polycylic aromatic hydrocarbons or radioactive particles, both groups of pollutants being of low irritant potency at low concentrations.

The weak effects of passive smoking were also seen in the multiple regression analyses. In children's FEV_1, there were some interactions between the number of smokers in the home and air conditioning, cooking fuel and heating respectively; the effects being strongest with one smoker versus zero smokers in the home rather than with two or more smokers versus one or zero smokers. The three way interactions added only slightly more to the effect on FEV_1. In the case-control part of this study, interaction between the use of gas stoves and the number of smokers in the home was absent.

Some unique sources of high RSP loading were found during the domestic air pollution sampling. In one home with a defective furnace, RSP levels of up to 460 ug/m^3 was found in the winter. In this residence, all four family members had chronic respiratory symptoms and decreased

lung function. In another home where kerosene lamps were used, the levels of NO_2 and RSP found were 218 and 265 ug/m^3 respectively. Many subjects were converting their heating systems to wood stoves to avoid dependency on oil and gas; there were no excesses in NO_2, SO_2 or RSP in these homes.

6.2 Outdoor Factors

In this study it was shown that the outdoor air pollution levels were significantly lower than the indoor levels, and since subjects tend to spend the majority of their time indoors, it is the more sensitive index to use to assess the relationship between air pollution and respiratory diseases. It is possible that in heavily industrialized cities the outdoor air pollution might be higher than indoors, but because of the percent of time indoors, outdoor levels may still not matter that much. In this study where air pollution levels were measured outdoors in all three towns, the average levels were low and the differences between the towns were significant, but differences in lung function and reporting of diseases between the town were absent or very small (Hosein et al, 1977a; Bouhuys et al 1978; Appendix 6.1). The air pollution levels indoors may remain at constant levels whereas the levels outside are always fluctuating because of spacial, temporal and climatic factors (Hosein et al 1977b). It is possible that residents who live very close to high polluting sources may be affected by the high air pollution levels outside, and probably by high levels inside which may occur because of outdoor-indoor diffusion. But in this study, when I looked at residents who lived closest to the air pollution sampling site with

the highest pollution levels, there was no evidence that these residents had lung function values and symptom reporting rates that were different from residents who lived in the residential areas with low air pollution levels (Appendix 6.2).

In the epidemiological studies to determine the causal factors of acute and chronic respiratory diseases, only in cases where the air pollution levels were extremely high were there reporting of excess diseases (Lawther et al, 1970; Carnow et al, 1969; Anderson et al 1965 a, b, c, and d; Holland et al, 1965; Colley et al, 1967; Neri et al 1975; Audry et al 1979). In the majority of cases, multiple regression and multivariate analyses were not conducted, and very often the effect of temperature was found to explain some of the differences between the residential groups.

6.3 Sources of Errors

6.3.1 Response Rates

The overall response rate in this study was 61.2%. The highest participation rate was in the children where in excess of 90% was tested in each town. In general the participation rate of adult females (61%) was better than males (58%); and the participation of whites (59%) was higher than blacks (48%), with the lowest participation rate being among black adult males (40%). In order to determine whether respondents were similar to non-respondents with regards to symptoms and smoking habits, a specific area in each town was chosen for a door to door survey. A

short questionnaire was applied covering questions on cough, wheeze, dyspnea, phlegm and smoking habits and history of previous lung diseases. The response rate from this follow-up was about 80%. In general there were some small differences in certain sub-groups by age, sex and race, but the number of significant differences was minimal and no consistent patterns emerged. These were some of the findings:

(a) In Lebanon and Ansonia, symptom prevalences in respondents and non-respondents were the same.

(b) Non respondent young males and females in Ansonia had a high percentage of smokers.

(c) Non-respondent women (45-64 years old) had more dyspnea than respondents in Lebanon.

(d) In white males from Winnsboro (45-64 years old) the non-respondents included a lower proportion of ex-smokers, but a higher proportion of smokers; there were no corresponding differences in symptoms prevalence.

(e) Black young males and females from Winnsboro who were non-respondents included a higher proportion of smokers, but only in black males was this accompanied with a higher proportion reporting phlegm.

6.3.2 Definition of Exposure

No attempts were made to elaborate on any of the domestic exposure questions. Consequently many assumptions were made about the exposure to each factor.

The frequency and duration of exposure were not determined for any of the factors. Nor was it determined whether personal lifestyle habits influenced the exposure factors, for instance:

(a) whether some subject did not keep pets for health reasons,

(b) whether humidifiers and air conditioners were acquired for health reasons,

(c) whether hot water heating was used primarily in the older homes in the lower income areas, and

(d) whether smokers smoked outside of the home for health or hygiene reasons.

6.3.3 Definition of Health Effects

The correlation between symptom response and lung function was not always strong, probably because, in some cases, the symptoms may not be severe enough to result in impairment of airways calibre. In other cases, there might be functional loss without any symptoms.

The correlation between the three lung function variable was also weak. This may be due to the fact that each variable measures the response from a different part of the lung. The FEV_1 is independent of effort and is a measure of mid-volume resistance. The $MF_{50\%}$ is determined by elastic recoil of the tissues and is a measure of small airways caliber. The $MF_{25\%}$ is determined by elastic recoil also and is a measure of airways resistance at low lung volumes. The intra-and

inter-subject variability in $MF_{50\%}$ and $MF_{25\%}$ between sequential measurements are normally high; the large standard deviation between these measures may reduce the likelihood of statistically significant differences between the means. In practice the standard deviations of FEV_1 measurements are small i.e. with a coefficient of variation of less than 5%.

6.3.4 Age of Test Groups

The main effects of domestic exposures were observed in children between 7-17 for males and 7-14 for females. Younger children may have been even more sensitive to test, but they are normally unable to perform the lung function tests satisfactorily and they may not be able to convey their symptoms in a consistent way. I tested the group between 7-11 to determine whether they were in any way different from the older children but no significant differences were found in the univariate and multiple regression analyses.

6.3.5 Measurement of Domestic Air Pollution

The device used in measuring the pollutants was well validated with other accepted methods. The sampler was too large though to be used as a personal sampler. Silverman et al (1982) found that the levels from personal exposures were generally different from indoor levels alone, but the correlation between both measures was high. I measured the exposure for one 24-hour period in the heating season and one 24-hour

period in the non-heating season; and although the interday variation was low, the estimation of exposure would have been more sensitive if more measurements were undertaken in each season. During the sampling days, no assessments were made regarding the duration of use of the stoves, the air conditioning units, and the number of cigarettes smoked.

6.3.6 Interviewer Variation

The chi-square contingency analysis was used to determine the variation in reporting of 14 key responses of the questionnaire. A summary of the findings are given here:

a. In Ansonia, interviewer variation occurred in only 5% of the comparisons made.

b. In Lebanon, one interviewer over-reported and one under-reported on the "usual cough" question, whereas in the other cough questions, there was no interviewer variation. Similar findings also occurred with the phlegm questions.

c. In Lebanon, there was some interviewer variation in some subgroups for the "ever wheeze" question, but no variation for the other wheeze questions or any of the dyspnea questions.

d. In Winnsboro, the analyses were conducted to determine whether there were any differences between the white and black interviewers. White males, aged 45-64, under-reported all symptoms to black interviewers, but there was no corresponding reporting in smoking habits. Younger white males showed no interviewer variation. Interviewer variation was absent when black subjects were interviewed.

6.4 Recommendations for Future Work

i. The questionnaire on domestic exposure factors should be expanded to include the duration and frequency of exposure.

ii. The actual number of cigarettes smoked indoors should be computed.

iii. The amount of dilution through leakage and exhaust should be estimated.

iv. The ways in which gas stoves can be ventilated directly to the outside should be investigated.

v. The relative humidity, age, diffusion rate and dust loading in hot water heated homes should be studied.

vi. The reason for acquiring air conditioning and humidifiers should be investigated.

vii. A better personal sampler should be developed to determine personal exposure over 24-hours per day over many days.

viii. Other pollutants should be measured e.g. formaldehyde, sulphates, nitrates and pollens.

ix. The causes of respiratory diseases in black children should be investigated since only the heating variable was consistent in explaining some of the changes.

x. A larger case/control population should be studied so that the power of detecting a real difference could be increased. The size of such a sample could be calculated if the standard deviation could be assessed, the size of the expected differences between the groups could be pre-determined, and the power could be chosen.

Many of these questions have been incorporated in follow-up work being conducted by this researcher in conjunction with other researchers at the Gage Research Institute, University of Toronto.

CHAPTER 7

CONCLUSION

In Section 1.2, I set out to determine the respiratory symptoms reporting rates and lung function of a group of residents from three townships, and to determine how symptoms and lung function were affected by nine domestic air pollution factors. I also undertook a case-control study and measured the air pollution exposure of cases and controls using a unique air pollution sampler developed specifically for sampling in homes, and to use the air pollution data to explain any health effects in the sub-group of residents and to determine whether the data validated the findings from the nine domestic factors.

The symptoms data, lung function measurements and domestic exposure data were collected on 4,074 non-smoking subjects. Air pollution measurements were undertaken in the homes of 26 cases and 26 controls, once in the winter and once in the summer.

The findings indicate that domestic pets, and the use of fireplaces and humidifers had no consistent effects on the reporting of respiratory symptoms or on lung function. Exposure to gases, vapours and dusts in the homes from hobbies, exposure to emissions from gas stoves, the absence of air conditioning, the use of hot water heating systems, domestic crowding, and the number of smokers in the home resulted generally in increased reporting of symptoms and decreased lung function, with the smokers variable showing the least effect, and the

heating variable, the greatest effect. Many of the exposure variables acted separately in influencing the reporting of symptoms and the changes in lung function.

There were consistent interactions between the exposure variables demonstrated by 2-way, 3-way, 4-way and 6-way comparisons with the cooking fuel, air conditioning and heating variables showing the largest effects on lung function. White children showed consistently the greatest responses to all of the exposure variables, whereas in the other groups, the hot water heating variable was most consistent. The effect on lung function was seen consistently in the FEV_1.

The air pollution study showed that indoor air pollution was higher than outdoors and that the use of gas stoves resulted in about 100 ug/m^3 more NO_2 in the home than the use of electric stoves. Cigarette smoking indoors resulted in higher suspended particulate matter when compared to homes where there was no smoking. NO_2 and RSP correlated separately with the lung function parameters. There were no significant differences in air pollution levels between the cases and controls. Subjects who lived in homes with gas stoves had lower lung function measurements than those who lived in homes with electric stoves.

APPENDIX 1.1 AIR POLLUTION AND CHRONIC DISEASES - LONGITUDINAL STUDIES

Study/Location	Characteristics	Findings	Comments
Anderson et al (1965a, 1965b, 1965c; 1965d) Chilliwack, B.C.	Valley town; primary occupation is dairying; no major air pollution sources; radio announced study; 5 sampling sites; 7 pollutants measured; modified MRC questionnaire; lung function tested; this town compared with Berlin, New Hampshire.	Total population was 8259; 3800 employed in pulp plant; 600 randomly selected; 95% participation; air pollution lower than Berlin; symptoms lower in Chilliwack; lung function better in Chilliwack; heart disease higher in Chilliwack.	Air pollution measures not very sensitive; population consisted of German extract, Berlin had more of English extract; there was observer variation; no nose clip used in lung function test.
Ferris et al (1964) Berlin, New Hampshire	Valley town; pulp mill in town; sulfite process changed in 1963; mill was major employer; radio announced study frequently; modified MRC questionnaire; lung function done with Benedic spirometer; air pollution measured at 3 stations.	Total population was 17,821; stratified sample chosen; function better in Chilliwack; symptoms less in Chilliwack.	Effect of occupation not considered.
Ferris et al (1971) Berlin, New Hampshire	More subjects added to previous group; demography of both groups differed slightly; 9 air pollution sites used; same questionnaire ; different observers; Stead-Wells spirometer.	There was a decrease in smoking and intensity of smoking; no observer variation; small improvement of function and symptoms; slight improvement of pollution.	Smoking could have accounted for some change; 79% of previous population used; small differences in spirometer readings.

APPENDIX 1.1 (CONT'D)

Ferris et al (1973), (1976) Berlin, New Hampshire	No more subjects added, some decrease because of emigration and death; air pollution data supplied by State; Stead-Wells spirometer used.	Slight improvement in air pollution; no more decrement in LF or sympton reporting.	Air pollution levels too low to be responsible for any physiologic changes; comparison is now a survivor population; air pollution methods changed.
Ferris et al (1979) Six US cities (No data available on follow-up)	Six-city study covering Mass., Tenn., Missouri, Ohio, Kansas and Wisconsin; one pollution site in each town; 5 pollutants monitored; adults only; NHLI questionnaire used; lung function tested.	Random selection; total selected was 18,079; total seen was 8480; air pollution highest in Missouri and Ohio towns; personal interview; there were no differences between cities for symptoms and lung function; SO_2 maximum was 110 ug/m^3; TSP was 180 ug/m^3.	One site in each town may not be good enough; less than 50% participation; no data on non-respondents; no data on why towns were chosen.
Sawicki (1977) Cracow, Poland	Dwellings randomly selected in Cracow; adults only; pollution measured but no data on no. of sites; study conducted in 1968 and repeated in 1973; interviewers trained; lung function performed on 70% of subjects.	Lung function was lower in 1973 but air pollution did not change significantly; levels for SO_2 = 50 ug/m^3; TSP = 80 ug/m^3; chronic bronchitis and asthma higher in 1973; 96% of 1968 population retested in 1973.	No data on work exposure; no standardization for aging; air pollution levels too low to create change.
Lebowitz et al (1975a and 1975b) Tucson, Arizona (no data available on follow-up)	White adults only; households chosen by stratification; multi-stage sampling; no air pollution data; NHLI questionnaire; lung function tests in clinic; nurse applied questionnaire.	No data given on lung function; good distribution in each stratum; no disease gradient between strata; 2989 in population 55% participated; no differences between participants and non-participants; migrants had more bronchitis and asthma.	Low participation rate; migrants to Arizona may be for health reasons; biased for age.

APPENDIX 1.2 AIR POLLUTION AND LUNG DISEASES - CROSS SECTIONAL STUDIES

Study/Location	Characteristics	Findings	Comments
Comstock et al (1973) Eastern Seaboard in the US	Workers selected to fit geographic distribution in Manhattan, Westchester, Baltimore, and Washington DC; telephone workers including one group from Japan; an air pollution station in each US city; MRC questionnaire used with trained interviewer; FEV_1 measured.	Age distribution different between US and Japanese i.e. 40-65 vs 40-60; study covered 3 separate time periods i.e. 1962 and 1968 in US and 1967 in Japan; men were selected; participation rate was variable and low; air pollution: Man> Balt> DC> West; symptom score: Man < West < Balt < DC.	Less than 50% participation; variable time period; inadequate air pollution data; age distribution different between cities; symptom scoring was very crude.
Holland et al (1969) Eastern Seaboard, US	Telephone workers chosen in 3 Eastern cities; Air pollution measured; MRC questionnaire applied by 8 observers.	No differences in air pollution between Washington DC, Westchester and Baltimore; no differences in symptoms; 97% response rate; US workers had less symptoms than UK workers.	Air pollution differed from Comstock; no observer variation despite 8 being used.
Stebbings (1971a, 1971b) Hagerstown, Maryland	White male residents who were non-smokers selected to determine urban-rural gradient in lung function and diseases; MRC questionnaire used; no pollution measured.	Age range between 35-64; 736 white males resident in Hagerstown but for 20 years lived outside the town; 578 interviewed; urban-rural gradient for symptoms and lung function.	Interviewer bias found; non-respondents were different from respondents in socio-economic status; professionals excluded; occupational exposure not considered; selective mobility.

APPENDIX 1.2 (CONT'D)

Winkelstein et al (1969) Buffalo, New York	21 sites selected for air pollution monitoring; stratified random sampling of households; age selected was 15+; short questionnaire; personal interview.	1369 chosen - 79% responded; correlation found between cough and phlegm with TSP in non-smokers over 45 years; no effect with SO_2.	study designed for blood pressure, but few questions asked on respiratory symptoms; poor air pollution methods; poor reproducibility on retest of 50 subjects; no data on sex and race distribution.
Mostardi et al (1974) Barberton and Revere, Ohio	Lifelong male group from each town studied; questionnaire applied; lung function tested; air pollution measured by Ohio State.	Revere was slightly lower than Barberton in air pollution, both usually less than 100 ug/m^3 for SO_2 and TSP; 50 males from Revere and 42 from Barberton were matched for age, weight, height; Revere residents had better FEV_1 and FVC; Revere had less infections.	No criteria given for group selection; very small sample, <100; symptom reporting based on reports by participants (young adults); air pollution differences too small to account for differences.
Petrilli et al (1966) Italy	Three towns were chosen to represent industrial, residential and suburban areas; females chosen who were never occupationally exposed and were 65+ years; MRC questionnaire applied.	The industrial town had more TSP and SO_2 than the residential or suburban; the suburban had lowest levels; more symptoms in industrial area than residential or suburban.	Data on previous residence not given; climate was different between areas.

APPENDIX 1.2 (CONT'D)

Aubry et al (1979) Quebec, Canada	Population selected in 3 areas of similar age and socio-economic class; subjects selected from tax list based on 3 year residence in area; air pollution supplied by Ministry of Environment, some data provided by McGill samplers; MRC-NHLI questionnaire administered.	Random samples from tax list; response rate low 55 to 60%; town B had more TSP but less SO_2 than town P; town S had less SO_2 and TSP than towns B and P; towns B and P had more cough and phlegm than S; town B had lower lung function than towns P and S.	Low response rate; follow-up rate was lower; non-respondents were different from respondents; use of different data bases for air pollution; no data on occupation given; generally air pollution was low with TSP 130ug/m^3 and SO_2 123 ug/m^3.
Nari et al (1975) Ottawa and Sudbury, Canada	Adult population chosen from each town using stratified sampling; air pollution data provided by Ministry of Environment who had 2 sites in Sudbury and one in Ottawa; MRC questionnaire applied; lung function test applied.	Ottawa group had 3631 participants and Sudbury had 2421; response rates 90%; FEV_1: FVC ratio used to assess differences; Ottawa had better ratios; Ottawa had lower symptom rates; SO_2 higher in Sudbury, but TSP higher in Ottawa; same climate; males had lower function than females.	Study of towns was 2½ years apart; occupation, smoking and age not considered in discussion; class may be different since one town in primarily blue collar, the other being white; interviewers consisted of nurses and volunteers resulting in interviewer bias.
Brooks et al (1972) London, England	Attendants to a public exhibition asked to participate; short questionnaire applied; peak flow rate measured.	2891 voluntered of which 2169 were males; no differences between smokers and non-smokers in PEFR up to age 45; residents of London had worst PEFR compared to previous London residents and non-Londoners.	Selective population, not very sensitive measure of function; biased by sex and class.

APPENDIX 1.2 (CONT'D)

Lawther et al (1970) London, England	Medical students requested to participate; MRC questionnaire applied; study repeated after 4 years; lung function tested.	Lung function increased over period; more students had cough and phlegm than previously.	Spirometer used in follow-up was different; no evidence of correcting lung function for age; bias because of training.
Reid (1958) London, England	Population selected from owners of single homes and with family size of 5 or more; nasal discharge monitored; symptoms assessed by visit to homes.	No association with home dampness; larger family size had more symptoms and nasal discharge.	Selected population, likely to be upper class; no data on participation size.
Holland et al (1965) London, England	Workers selected in London and suburbs; MRC questionnaire applied; lung function measured; sputum samples collected; no air pollution data.	293 mailmen from London, 477 from suburbs; no differences between groups for symptoms; PEFR and FEV_1 better in suburb workers; more sputum in London workers.	Small populations not identical occupation; no accounting for smoking or migration.
Colley et al (1967) London, England (children followed-up, see Table 2.3 - Holland et al, 1969b)	Two residential areas chosen, in SW and NE London; MRC questionnaire applied; peak flow determined; interview staff trained.	90% of persons responded to study; no differences between areas; morning cough showed a social class gradient in fathers; 1/3 of sample re-visited to check validity and response was good; no differences in PEFR.	NE London had more residents of lower social class; work exposure and smoking not considered; no pollution data given.

APPENDIX 1.2 (CONT'D)

Cohen et al (1972) **San Gabriel and San Diego, California**	Members of Seventh Day Adventist Church chosen in San Diego and San Gabriel; whites only; non-smokers; all on church list; 6 churches selected in San Gabriel, 4 in San Diego; MRC questionnaire applied; studied in winter to minimize acute ozone effects; air pollution data from State stations.	San Gabriel had more ozone than San Diego; 82% tested in San Diego, 67% in San Gabriel; no differences between towns for lung function or symptoms; overall population has less than 2% of chronic bronchitis; similar meteorlogically.	Very selective population; no consideration for non-participants; previous urban residents were excluded from San Diego population but not San Gabriel; bias because of group who are interested in health; occupation not covered.
Linn et al (1976) **San Francisco and Los Angeles, California**	Office workers were studied who worked in San Francisco (SF) and Los Angeles (LA); groups matched for age, sex and residential history; air pollution data provided by Cities; NHLI questionnaire applied; lung function tested.	LA had more ozone, NO_2 and TSP than SF; females in LA had more cough and phlegm than SF; no differences between cities for lung function; LA had 206 participants, SF had 441; 95% responded.	Ozone levels less than 160 ug/m^3 in each city, not high enough to cause differences; worker population usually healthier than general population; racial and language differences not assessed; no standardization for smoking.
Detels et al (1979) **Tashkin et al (1979)** **Burbank and Lancaster, California**	Households were chosen in Burbank (B) and Lancaster (L); both group matched for race, age, sex; NHLI questionnaire applied; lung function tested; no air pollution data given.	Burbank said to have more O_3 and other pollutants than Lancaster; Burbank had depressed FEV_1, $MEF_{50\%}$ and $MEF_{25\%}$; Lancaster had more symptoms; 2134 households identified in Lancaster-79% tested; 91% household identified in Burbank - 70% tested.	No good data on exposures; no evaluation of occupational exposures; no data on previous residence; no account taken of non-participation.

APPENDIX 2.1 AIR POLLUTION AND CHILDHOOD DISEASES - LONGITUDINAL STUDIES

Study/Location	Characteristics	Findings	Comments
Lunn et al (1967) Sheffield, England	Four areas chosen in Sheffield with differing pollution and housing; pollution data supplied by cit , some data generated by researchers; 5 year old selected; questionnaire applied to parents; lung function tested; study done in summer 1964.	Town A had highest SO_2 and TSP and lowest quality housing; nasal discharge high in town A; more cough and infection in town A; bronchitis and pneumonia also highest; lung function was lowest in town A; pollution said to be more important than social class.	Domestic exposure not considered; small group in town A (82); lung function manoeuver may be variable in the young.
Lunn et al (1970) Sheffield, England (Follow-up)	Repeated in summer, 1968; air pollution measured as before.	Those with previous illness had higher risk of getting it again; areal difference disappeared in lung function and symptoms.	No data given on number loss to follow-up.
Leeder et al (1974, 1976 and 1977) New South Wales, Australia	All schools in 24 km radius of Sydney chosen; selection based on multi-stage sampling; questionnaire completed by mother; lung function tested; no air pollution data; questionnaire validated by re-visiting 200 homes.	6000 in 7-8 yr. group; 6000 in 12-13 year group; more asthma and bronchitis in boys than girls; parents with many children reported more cough and phlegm than smaller families; children with diseased parents had more diseases; parental smoking had small effect.	Parental questionnaire on children; no reasons given for high disease rates in children.

APPENDIX 2.1 (CONT'D)

Peat et al (1980a, 1980b) (Follow-up of Leeder et al, study in New South Wales, Australia)	All schools in 24 km radius of Sydney chosen; selection based on multi-stage sampling; questionnaire completed by mother; lung function tested; no air pollution data; questionnaire validated by re-visiting 200 homes.	Boys still had more asthma (11.5% vs 4.2%) and bronchitis (24% vs 13.9%) than girls; some decrease in rate over time; no relation between acute infection and family size; more bronchitis and less asthma in suburbs; more disease in native people.	No data on migration; no data on air pollution; no reasons given for observations.
Douglas et al (1966) England and Wales	Children born during one week in March 1946; air pollution based on coal consumption in 2689 areas; four areas created from A to D i.e. low to high pollution; mothers were questionned about children's illness.	Total born = 13,687; illegitimate twins, professionals, agricultural excluded; sample of 5362, of whom 4592 were living in UK in 1961; lower respiratory tract infection increased with pollution loading in boys and girls for middle and lower class families, and lasted for 15 years.	Very selective population; about 60% excluded; air pollution index crude.
Colley et al (1973) (Follow-up at age 20 years)	Children born during one week in March 1946; air pollution based on coal consumption in 2689 areas; four areas created from A to D, i.e. low to high pollution; mothers were questionned about children's illness. MRC questionnaire applied directly to young adults.	3899 participated in follow-up; effect of smoking most predominant over air pollution and social class; children with cough at 2 years showed more illness than young adults.	Different assessment method used; smaller population; no pollution data.

APPENDIX 2.1 (CONT'D)

Kiernan et al (1976) (Follow-up at age 25 yrs)	Children born during one week in March 1946; air pollution based on coal consumption in 2689 areas; four areas created from A to D, i.e. low to high pollution; mothers were questionned about children's illness; MRC questionnaire applied directly to young adults.	3245 replied of which 2088 were analysed; childhood illness still important in adult illness; smoking more important than social class and air pollution.	Smaller population; no pollution data.
Irvine et al (1980) London, England	Group selected from London and Crawley; no data on selection criteria except they were born just prior to London 1952 fog; MRC questionnaire applied; lung function tested; no air pollution data.	3000 selected; smoking most important factor; no differences in FEV_1 between areas; more symptoms in London; previous childhood illness important in adult disease.	Not sufficient data on selection criteria.

APPENDIX 2.2 AIR POLLUTION AND CHILDHOOD DISEASES - CROSS SECTIONAL STUDIES

Study/Location	Characteristics	Findings	Comments
Lebovitz et al (1974) Tucson, Arizona	School children who exercised in polluted and un-polluted areas; air pollution samplers placed within gym in Tucson, outdoor in Tucson, and outdoors in copper smelting town; lung function tested.	Indoor group showed no post-exercise effect in lung function; outdoor group in Tucson showed post-exercise effect on high pollution days; outdoor group in smelting town showed post-exercise decrease on high pollution days.	Inadequate pollution data provided by State; small numbers in each group; the smelting town had more Mexican-American.
Shy et al (1970) Chattanooga, Tennessee	Second grade children were selected in two areas, one close to a NO_2 source; questionnaire applied to parents.	4,043 chosen; differences between areas for illness, highest being in area with higher NO_2.	Data complicated by two bouts of influenza; incomplete data on population selection and exposure.
Shy et al (1973) 3 Cities, US	Children chosen from Chattanooga, Cincinatti, and New York; lung function tested.	987 from 2nd grade, Chattanooga; 394 from 2nd grade, Cincinatti; 2364 from grades 1-6, New York; reduced FEV_1 associated with high TSP and NO_2. No effects of NO_2 seen.	No data on how population selected; no details on pollution measurements; in general pollution was too low; social class not considered.

APPENDIX 2.2 (CONT'D)

McMillan et al (1969) Los Angeles, California	Two schools chosen in two areas 17 miles apart; population demographically similar; peak flow measured; air pollution data provided by LA city.	City A had more TSP but less NO_2 than city B; both had same SO_2; A had more oxidant; overall PEFR in A was higher than B.	Air pollution levels low for both areas therefore effects not expected; PEFR not sensitive to determine effect on small airways; 2 flow meters used but no data given on similarities; small population groups (50) and poor response rate (A = 80%, B = 50%).
Stebbings et al (1976a and 1976b) Pittsburgh, Pennsylvania	Air pollution episode evaluated in 4 schools in Pittsburgh and 2 schools from control district; lung function measured by one technician; air pollution provided by the State.	Approximately 50 children per school tested each morning; TSP was said to be 250 ug/m^3 and SO_4 26 ug/m^3; no effects were seen between groups.	Small population; episode not high enough to create changes; children with disease may have stayed away.
Paccagnella et al (1969) Ferrara, Italy	Four zones were chosen; one urban and 3 residential; air pollution was measured on top of each school; classes were chosen with age range of 7-12 years; medical assistant evaluated onset of illness.	The air pollution was highest in the urban town, but this area had highest social class; correlation found between illness and SO_2 and TSP.	No inference to O_3 or NO_2 as cause of association; no data on why areas were chosen.
Kagawa et al (1974, 1975) Tokyo, Japan	School selected because of willingness to participate; 10 males and 10 females; air pollution data available; lung function tested.	Correlation found between function and temperature; ozone was correlated with raw suggestion of synergism.	Small population; health status of participants not given; pollution levels low.

APPENDIX 2.2 (CONT'D).

Barrebijn et al (1975) Holland	Two areas selected with differing air pollution; 5th and 6th grades chosen; air pollution provided; 5 year residence criteria used; questionnaire was self administered; lung function tested.	Area I had 2104 children, area II had 276; participation was 96 and 97% respectively; both areas had similar rates for wheezing and past symptoms; Area II had less cough; Area I and II had same lung function; Area I had more SO_2 and smoke with maximum of 280 ug/m^3 and 60 ug/m^3 respectively.	Self administered questionnaire may produce bias; wide difference in population sizes; NO_2 not checked despite the presence of gas heated greenhouses in areas.
Holland et al (1969a) Kent, England	Four areas in Kent selected for study of children of ages 5, 11 and 14 years; peak flow performed by medical visitors; parents completed questionnaire; air pollution measured.	10,721 children seen; 94% studied; air pollution was as follows- Area 1>Area 2>Area 3>Area 4.; peak flow at area 1<area 2<area 3<area 4; social class effect seen with class 1 having highest PEFR; large families had lowest PEFR.	Inadequate measure of function; pollution level low; 12 observers used and variation was large; SO_2 not given, just stated; social class confounding.
Holland et al (1969b) Harrow, England	All families with babies from July 1963 to June 1965 included; questionnaire applied to parents; 2 areas studied; air pollution data given for both areas; peak flow measured.	2365 families in study, 93% participated; area 1 had marginally more SO_2 and TSP than area 2; no areal difference for father symptoms, but present for mother; no observer bias; validation test of 1/3 population showed minor bias; children in area 1 had more cough; no data on peak flow.	PEFR not sensitive test.

APPENDIX 2.2 (CONT'D)

Colley et al (1970) England and Wales	500 children were chosen in each sex, each year in each of 6 areas in the age range of 6-10 years; conducted at end of summer; smoke pollution data available; peak flow measured; questionnaire filled out by mother.	11,135 selected, 98% studied; cough was associated with past history of infe[illegible] gradient with air pollution and symptoms; no gradient with peak flow.	Peak flow test insensitive; air pollution low; observer variation high on use of PEFR.
Colley et al (1974) Buckinghamshire, England	Area selected in Buckinghamshire, 7 schools included with ages 6 to 14 years; self administered questionnaire; no pollution data.	2598 children selected, 93% participated; parental smoking showed small (not significant) gradient; suggestion of parental disease from smoking leading to parental infection then to children infection.	Self administered questionnaire subject to bias; no validation of questionnaire..

THE FACTS

ANSONIA WARD 4 BREATHING STUDY

Who is conducting the Test? The Yale Lung Research Center under the direction of Dr. Arend Bouhuys and Dr. Charles Mitchell.

Why is the Test necessary? So we can determine what effect air pollution has on your breathing capacity.

Why have I been chosen? You live in a high air pollution area.

Who will be Tested? All Ansonia residents of Ward 4, seven years of age and older.

When will the Test be administered? A volunteer will visit you in your home and discuss the time and date of your breathing test.

Where is the Test done? There is no physical examination or needles. A confidential 10 minute interview will be conducted followed by a simple breathing test. The test will be given in a specially designed trailer located at the Pine School.

How much does it cost? There is no charge to you and your family. The test is being conducted through a contract with the National Heart and Lung Institute, National Institutes of Health, Department of Health, Education and Welfare.

How can I help? You can help by being tested and insuring that each member of your family is tested. Without **you** our test results will not be accurate.

By the way — who is sponsoring this program?

Yale University School of Medicine
Lower Naugatuck Valley Community Council
Valley Health Department
Griffin Hospital
Ansonia Public School System
Local Industries
Tuberculosis and Respiratory Disease Association of South Central Connecticut

THE FACTS

WINNSBORO BREATHING STUDY

WHO IS CONDUCTING THE TEST? The Yale Lung Research Center under the direction of Dr. Arend Bouhuys and Dr. Charles Mitchell.

WHY IS THE TEST NECESSARY? So we can determine whether low air pollution has any effect on your breathing capacity.

WHY HAS WINNSBORO BEEN CHOSEN? Because it is a low air pollution area in the South.

WHO WILL BE TESTED? All residents of Winnsboro, seven years of age and older.

WHEN WILL THE TEST BE ADMINISTERED? A volunteer will visit you in your home and discuss the time and date of your breathing test.

WHERE IS THE TEST DONE? There is NO physical examination or needles. A confidential 10 minute interview will be conducted followed by a simple breathing test. The test will be given in a specially designed trailer located at various schools around town.

HOW MUCH DOES IT COST? There is no charge to you and your family. The test is being conducted through a contract with the National Heart and Lung Institute, National Institutes of Health, Department of Health, Education and Welfare.

HOW CAN YOU HELP? You can help by being tested and ensuring that each member of your family is tested. Without YOU our test results will not be accurate.

WHAT HAPPENS TO YOUR RESULTS? Your individual results are reviewed by a physician. If any abnormality is found, both you and your own physician will be notified.

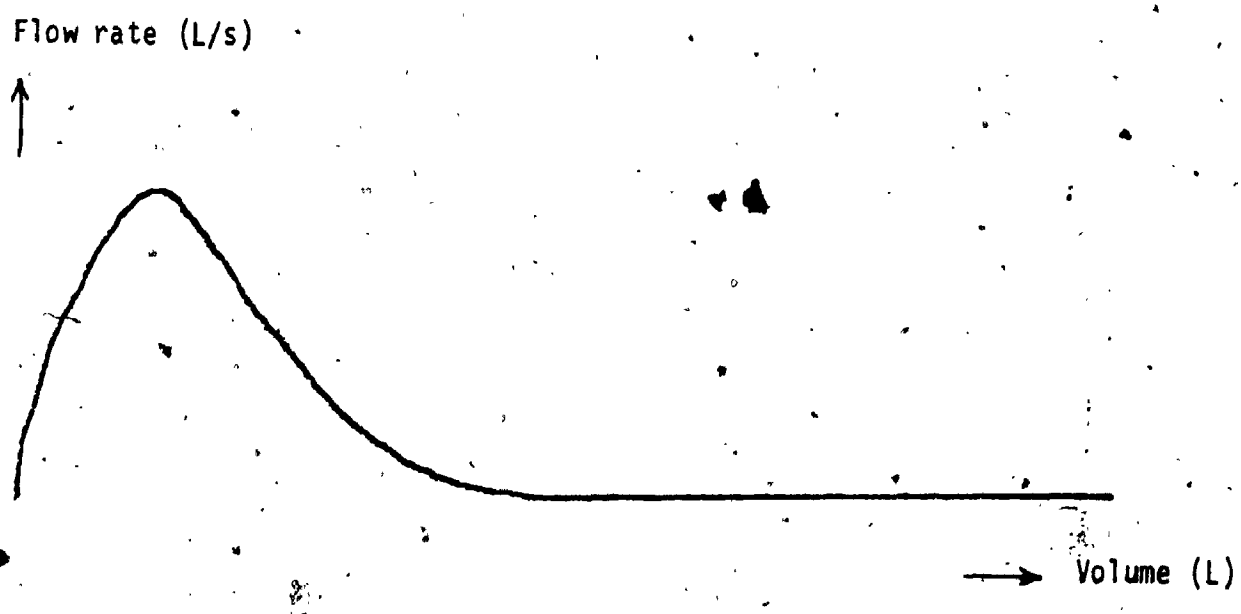
BY THE WAY -- WHO IS SPONSORING THIS PROGRAM?

...Yale University School of Medicine
...Town of Winnsboro
...Fairfield County Council
...Fairfield Memorial Hospital
...Winnsboro Chamber of Commerce and local industries
...South Carolina Department of Health and Environmental Control
...South Carolina Lung Association (formerly S.C. TB-RD Association)
...Fairfield County Department of Education
...Gordon Elementary School
...Everett Grammar School
...Mount Zion Elementary School
...Fairfield Jr. High School
...Winnsboro High School
...Richard Winn Academy

APPENDIX 3.3

HARD-COPY OUTPUT FROM COMPUTER AIDED LUNG FUNCTION TEST

```
FVC:      4.653 PERCENT:   105.502 PRED:     4.325
FEV1:     2.868 PERCENT:    83.232 PRED:     3.445 AS % FVC =   62.842
FEV3:     4.062 AS % FVC =   89.013
PEFR:     5.940 PERCENT:    59.703 PRED:     9.949
MF50:     3.153 PERCENT:    84.657 PRED:     3.724
MF25:      .760 COVAR:   -- 7.363
 19.0 13.0 26.0 120172 1492 3 10  182  110   51 032521
1 1 2 2 0 0 2 2 0 0 0 0 2 1 1 18 4 5 2 1 1
2 2 2 1 2 2 2 2 0 0 2 0 3 1 3 1 1 0 0 0 0
0 0 0 0 0 0 2 1 2 2 2 2 2 2 2 2 2 2 2 2 2
2 1 2 2 0 0 0 0 0 0 0 0 0 3 0 0 0 0 0 3 0
0 0 0 0     .0 1 1 49 1 0 0 0 0 0 1 1 1 1 1 1
7 1 1 2 1 7 2 3 2 2 1 1 2 4 1 2 2 1 2
ADDITIONAL DATA? N
```



LUNG RESEARCH CENTER QUESTIONNAIRE

1 Subject number

2 Subject category (coded to classify place of residence)

3 Interviewer number

4 Height (to nearest cm)

5 Weight (to nearest kg)

6 How old are you? (age at last birthday)

7 What is your birthdate? (enter 6 digits) ( ) ( ) month ( ) ( ) day ( ) ( ) year

8 Sex — 1. male 2. female

9 Race — 1. Caucasian 2. Negro 3. other

I SHALL NOW ASK YOU SOME QUESTIONS ABOUT COUGH:

14 Do you *usually* cough first thing in the morning? — 1. yes 2. no

15 Do you *usually* cough at other times during the day or night? — 1. yes 2. no
(If YES to 14 or 15, ask 16 and 17.)

16 Do you cough on most days for as much as 3 months of the year? — 1. yes 2. no

17 For how many years have you had this cough? — 1. less than 2 years 2. 2–5 years 3. more than 5 years

I SHALL NOW ASK YOU SOME QUESTIONS ABOUT PHLEGM, SPUTUM, OR MUCUS THAT COMES FROM YOUR CHEST:

20 Do you *usually* bring up (phlegm), (sputum), or (mucus) from your chest first thing in the morning? — 1. yes 2. no

21 Do you *usually* bring up (phlegm), (sputum), or (mucus) from your chest at other times during the day or night? — 1. yes 2. no
(If YES to 20 or 21, ask 22–25.)

22 Do you bring up (phlegm), (sputum), or (mucus) from your chest on most days for as much as 3 months of the year? — 1. yes 2. no

23 For how many years have you raised (phlegm), (sputum), or (mucus) from your chest? — 1. less than 2 years 2. 2–5 years 3. more than 5 years

24 What is the usual color of the (phlegm), (sputum), or (mucus) you bring up from your chest? — 1. don't know 2. clear 3. white 4. other 5. yellow 6. green
(If 1, 2, 3, or 4 ask 25.)

25 Have you ever had episodes of yellow or green (phlegm), (sputum), or (mucus)? — 1. yes 2. no

26 Have you ever coughed up blood? — 1. yes 2. no (specify)

I SHALL NOW ASK YOU SOME QUESTIONS ABOUT WHEEZING, WHISTLING, AND CHEST TIGHTNESS:

30 Have you ever noticed any wheezing, whistling, or tightness in your chest? — 1. yes 2. no
(If YES to 30, ask 31 to 53.)

31 Which of these symptoms have you experienced, wheezing or tightness or both?
1. only wheezing and whistling
2. only chest tightness
3. mainly wheezing and whistling
4. mainly chest tightness
5. both wheezing and whistling and chest tightness

32 At what age did your (wheezing), (whistling), or (chest tightness) first occur? ( ) ( )

33 When did this (wheezing), (whistling), or (chest tightness) last occur?
1. within last 4 weeks
2. within last 12 months
3. more than 1 year ago but less than 5 years ago
4. more than 5 years ago

34 How frequently have you experienced this (wheezing), (whistling), or (chest tightness)?
1. usually at least once a day or night
2. only a few times each week
3. only a few times each month
4. only a few times each year
5. only a few times ever
6. only once

(If answer 5 or 6 to 34, skip to 56.)

35 (Does) or (did) your (wheezing), (whistling), or (chest tightness) occur with colds or sore throats? 1. yes 2. no

36 (Does) or (did) your (wheezing), (whistling), or (chest tightness) occur with episodes of increased phlegm from your chest? 1. yes 2. no

37 (Is) or (was) your (wheezing), (whistling), or (chest tightness) associated with attacks of shortness of breath? 1. yes 2. no

(Is) or (was) your (wheezing), (whistling), or (chest tightness) brought on by, or made worse by exposure to:

38 House dust? 1. yes 2. no

39 Other dusts or fumes in the home? 1. yes 2. no (specify)

40 Contact with animals? 1. yes 2. no (specify)

41 Plants or pollens? 1. yes 2. no (specify)

42 Dusts, gases, or fumes at work? 1. yes 2. no (specify)

43 Tobacco smoke? 1. yes 2. no

44 Other factors? 1. yes 2. no (specify)

45 (Is) or (was) your (wheezing), (whistling), or (chest tightness) worse on any particular day or days of the week? In other words, is there any difference between, say, Friday, Monday, Sunday, or Thursday? 1. yes 2. no

(If YES to 45, ask 46 and 47.)

46 On which day or days is it worse?
1. first day back at work
2. other day(s) at work
3. weekends

47 Did this worsening occur sometimes or always? 1. sometimes 2. always

48 (Is) or (was) your (wheezing), (whistling), or (chest tightness) *better* on any particular day or days of the week or weekend? — 1. yes 2. no

(IF YES to 48, ask 49.)

49 When is it better? — 1. weekday 2. weekend

50 (Is) or (was) your (wheezing), (whistling), or (chest tightness) better, the same, or worse on vacation? — 1. better 2. the same 3. worse

51 (Is) or (was) your (wheezing), (whistling), or (chest tightness) *worse* during a particular season? — 1. yes 2. no

(If YES to 51, ask 52 and 53.)

52 Which (is) or (was) the worst season? — 1. winter 2. spring 3. summer 4. fall

53 (Do) or (did) these symptoms occur *only* during this season? — 1. yes 2. no

I SHALL NOW ASK YOU SOME QUESTIONS ABOUT BREATHLESSNESS:

56 Are you disabled by any condition, other than lung disease, which would interfere with your walking? — 1. yes 2. no (specify)

(If YES, go to 60.)

57 Are you troubled by shortness of breath when hurrying on level ground or walking up a slight hill? — 1. yes 2. no

(If NO, go to 60.)

58 Do you get short of breath walking with other people of your own age on level ground? — 1. yes 2. no

(If NO, go to 60.)

59 Do you get short of breath on walking ¼ mile on level ground in about 15 minutes? — 1. yes 2. no

(Dyspnea grades: 0 = no to #57; 1 = yes to #57; no to #58; 2 = yes to #58; no to #59; 3 = yes to #59)

NOW SOME QUESTIONS ABOUT CHEST ILLNESS:

60 During the past 3 years, how much trouble have you had with such illnesses as chest colds, bronchitis, or pneumonia? — 1. none 2. slight 3. some 4. considerable 5. a great deal

Have you ever had:

70 Bronchial asthma? — 1. yes 2. no

71 Bronchitis? — 1. yes 2. no

72 Pneumonia? — 1. yes 2. no

73 Pleurisy? — 1. yes 2. no

74 Pulmonary tuberculosis? — 1. yes 2. no

75 A chest injury, such as a fractured rib or spine? — 1. yes 2. no (specify)

76 A chest operation? — 1. yes 2. no (specify)

NOW, SOME QUESTIONS ABOUT SMOKING:

86	Do you *now* smoke cigarettes? (If NO, skip to 93.)	1. yes 2. no
87	Do you smoke cigarettes with or without filters?	1. with filters 2. without filters 3. both with and without filters
88	Do you inhale?	1. yes 2. no
89	How old were you when you began to smoke cigarettes?	____age
90	How many cigarettes do you usually smoke each day at the present time?	____number per day
91	At what age did you start smoking this many?	____age
92	Prior to this, how many did you smoke each day?	____number per day
	(Repeat 91 and 92 until total smoking history has been described.) (Skip to 102.)	
93	Did you *ever* smoke cigarettes? (If NO, skip to 102.)	1. yes 2. no
94	Did you smoke cigarettes with or without filters?	1. with filters 2. without filters 3. both with and without filters
95	Did you inhale?	1. yes 2. no
96	How old were you when you began to smoke cigarettes?	____age
97	How old were you when you stopped smoking cigarettes regularly?	____age
98	What was the usual number of cigarettes you smoked per day just before you stopped?	____number per day
99	At what age did you start smoking this many?	____age
100	Prior to this how many did you smoke each day?	____number per day
	(Repeat 99 and 100 until total smoking history has been described.)	
101	Were you influenced to stop because you had a cough, wheezing, or shortness of breath?	1. yes 2. no
102	Do you *now* smoke pipes or cigars? (If NO, skip to 106.)	1. yes 2. no
103	How many pipefuls or cigars do you usually smoke each day?	____number per day
104	Do you usually inhale when you smoke either pipes or cigars?	1. yes 2. no
105	How old were you when you first smoked pipes or cigars? (Skip to 112.)	____age
106	Did you *ever* smoke pipes or cigars? (If NO, skip to 112).	1. yes 2. no
107	How many pipefuls or cigars did you usually smoke each day?	____number per day
108	Did you usually inhale when you smoked either pipes or cigars?	1. yes 2. no
109	How old were you when you first smoked pipes or cigars?	____age
110	How old were you when you stopped smoking pipes or cigars?	____age
111	Were you influenced to stop because you had a cough, wheezing, or shortness of breath?	1. yes 2. no

NOW SOME QUESTIONS ABOUT SPECIFIC OCCUPATIONS:

Have you ever worked:

Answers below apply to every question from 112 to 120.

1. no
2. for less than 3 months
3. for 4 months to 1 year
4. for more than 1–5 years
5. for more than 5–10 years
6. for more than 10–20 years
7. for more than 20 years

112 At a coal mine?

113 In any other mine?

114 In a quarry?

115 In a foundry?

116 In a pottery?

117 In a cotton, flax, or hemp mill?

118 With asbestos?

119 On a farm?

(If YES, specify type of farm.)

120 In any other job with exposure to dust, gas, or fumes?

(If YES, specify type of exposure.)

I WOULD NOW LIKE TO ASK YOU SOME QUESTIONS ABOUT YOUR HOME AND HOBBIES:

121 Do you or does any member of your household keep animals or pets? 1. yes 2. no (specify)

122 Have you any hobby that exposes you to dusts, gases, or fumes, such as from paints, glues, or wood dust? 1. yes 2. no (specify)

123 Have you ever lived in any town or area other than here? 1. yes 2. no

(If YES, ask 124 and 125.)

124 At what age did you move into this town or area? ( ) ( )

125 Where did you live previously?
1. mainly country
2. mainly city
3. country and city

126 What type of heating system is used in your home?
1. none
2. forced air heating
3. circulating hot water or steam
4. electric radiant heating
5. other (specify)

127 In addition to the above, do you use a fireplace in your home? 1. yes 2. no

128 What fuel is used for cooking?
1. electricity
2. gas
3. wood
4. coal

129 Do you have a humidifier? 1. yes 2. no

130 Do you have air-conditioning? 1. yes 2. no

131 Do you have an air-cleaning device in the home? 1. yes 2. no

132 What is the number of people in your household? ( ) ( )

133 How many are smokers? ( ) ( )

APPENDIX 3.5

HOME QUESTIONNAIRE FOR CHILDREN AGED 9-15 YEARS

Name of Child:_____ School:

Birthdate:_____ Grade:
Month Day Year

1.	Has he/she ever had any wheezing, whistling or tightness in the chest?	YES [1] NO [2]	21
	If NO, proceed to question 8 *If YES, continue with question 2*		
2.	At what age did it first occur?	[ ] years	23
3.	When did this wheezing, whistling or tightness last occur?	Within last 4 weeks [1] Within last 12 months [2] More than one year ago but less than 5 years ago [3] More than 5 years ago [4]	24
4.	How frequently does or did he/she experience this whistling, wheezing or tightness?	Usually at least once a day or night [1] Only a few times each week [2] Only a few times each month [3] Only a few times each year [4] Only a few times ever [5] Only once [6]	25
	If answer 5 or 6, proceed to question 8 otherwise (i.e., answer 1-4) continue with question 5		
5.	Is it worse during a particular season?	YES [1] NO [2]	42
	If NO, proceed to question 8 *If YES, answer question 6 & 7*		
6.	Which is the worst season? (check only one season)	Winter . . . . . [1] Spring . . . . . [2] Summer . . . . . [3] Fall . . . . . [4]	43
7.	Do these symptoms occur only during this season?	YES [1] NO [2]	44
8.	Has he/she ever had episodes of sneezing and "runny nose?"	YES [1] NO [2]	76
	If NO, proceed to question 13 *If YES, continue with question 9*		
9.	How frequently does he/she experience these nose symptoms?	Usually at least once a day or night [1] Only a few times each week [2] Only a few times each month [3] Only a few times each year [4] Only a few times ever [5]	77
	If answer 5, proceed to question 13 otherwise (i.e., answers 1-4) continue with question 10.		
10.	Are these symptoms worse during a particular season?	YES [1] NO [2]	83
	If NO, proceed to question 13 *If YES, answer questions 11 & 12*		

11.	Which is the worst season? (check only one season)	Winter . . . . . [1] Spring . . . . . [2] Summer . . . . . [3] Fall . . . . . [4]	84
12.	Do these symptoms occur only during this season?	YES [1] NO [2]	85
13.	Has he/she ever lived in any town or area other than here? *If NO, proceed to question 16* *If YES, answer questions 14 & 15*	YES [1] NO [2]	121
14.	At what age did he/she move into this town?	[ ] years	122
15.	Where did he/she live previously?	Mainly country . . . . . [1] Mainly city . . . . . [2] Country and city . . . . . [3]	123
16.	What type of heating system is used in your home?	None [1] Forced air heating [2] Circulating hot water or steam [3] Electric radiant heating [4] Other - specify _____ [5]	124
17.	In addition to the above do you ever use a fireplace in your home?	YES [1] NO [2]	125
18.	What fuel is used for cooking?	Electricity . . . . . [1] Gas . . . . . [2] Wood . . . . . [3] Coal . . . . . [4]	126
19.	Do you have humidifiers or a humidification system?	YES [1] NO [2]	127
20.	Do you have air-conditioning?	YES [1] NO [2]	128
21.	Do you have an air cleaning device in the home?	YES [1] NO [2]	129
22.	What is the number of people in your household?	[ ]	130
23.	How many are smokers? (5 or more cigarettes a day)	[ ]	131

FAMILY HISTORY

Have you (the parent), your spouse or any of your other children ever had:

24.	Asthma?	YES [1] NO [2]	132
25.	Any kind of chronic bronchial trouble?	YES [1] NO [2]	133
26.	Other lung disease?	YES [1] NO [2]	134
27.	Hay fever or similar nose allergies?	YES [1] NO [2]	135

Thank you, that is the end of the questionnaire. Please return it with your signature to indicate consent to your child having the test at school.

Signature _____

APPENDIX 3.6

PERSONAL ENVIRONMENT MONITOR-INSTRUCTIONS TO SUBJECTS

LUNG RESEARCH CENTER

YALE MEDICAL SCHOOL

1. Always keep suitcase upright. If tilted solutions inside may spill and clog samplers.

2. When at home or in office keep line plugged into power source.

3. Keep sampler on chair or table so it will be at the same height as your nostrils, e.g. when you are up and about, keep on table and when lying down, keep on chair.

4. If the noise becomes too disturbing, then keep the sampler in the next closest position that is not disturbing.

5. Always keep sampler running. In case of emergency, turn off and unplug.

Call Roland Hosein

Office - 432-4430
Home - 624-5781

Thank you.

APPENDIX 3.7

FOLLOW-UP QUESTIONNAIRE FOR DOMESTIC STUDY

GROUP: _____

NAME: _____ CODE: _____

ADDRESS: _____ TOWN: _____

TELEPHONE: _____ AGE: _____

SYMPTOMS: _____ OCCUPATION: _____

SMOKING HABIT: _____ WHERE: _____

HEATING: _____ FIREPLACE: _____ A/C: _____

COOKING: _____

HOUSEKEEPING: _____ SIZE OF HOUSE: _____

FAMILY SIZE: _____ SIZE OF SAMPLING AREA: _____

FAMILY SYMPTOMS: _____ HOW MANY SMOKERS? _____

FEV_1/FVC: _____ CARPETED? _____

$MF_{50\%}$ _____

	PES		OES	
	Winter	Summer	Winter	Summer
SO_2				
NO_2				
RSP				

WEATHER t_{air} t_{wet} velocity direction RH Sky Condition

WINTER _____

SUMMER _____

APPENDIX 3.8

SAMPLING AND ANALYTICAL METHODS FOR DOMESTIC AIR POLLUTION

i. Selection of Filter Type:

Filters of polyvinyl chloride (PVC) membrane, cellulose nitrate (CN) membrane, and glass fiber (GF) were tested for use in the cyclone sampler. Four filters of each type were subjected to two methods of conditioning: first, they were submitted for 24-hour periods to a constant relative humidity (RH) of 50% or [illegible]% at a constant temperature (25°C); second, they were desiccated in an oven at 45°C for four days continuously and weighed at 24-hour intervals in a Cahn Microbalance Mod[illegible] 4100 (Ventron Corp.). Glass fiber filters were found to have the best characteristics for particulate sampling in the domestic environment

ii. Calibration:

Two personal environment samplers (PES) were built and were compared with one another and with standard equipment to determine the sampling efficiency for each pollutant. All comparisons were made over 24-hour periods in low and high pollution concentrations. For airborne particulate matter, the standard equipment u[illegible]d is the high-volume air sampler adapted with a cascade impactor, the collection medium being glass-fiber filters (Gussman et al, 1973). This sampler assembly has the same collection characteristics and size cutoff as the 10 mm. nylon

respirable-particulate sampler (Gussman et al, 1973). For collecting NO_2 and SO_2, the Research Appliance Company (RAC) bubbling train assembly was used.

iii. Analyses:

Analyses for SO_2 and NO_2 were conducted according to standardized methods.

a) Sulphur Dioxide (West and Gaeke, 1956; Environmental Protection Agency, 1971).

Collection:

Air was sampled through a manifold at about 1 l.p.m. One stream (0.2 l.p.m.) led to an impinger containing a solution (50 ml.) of 0.04M potassium tetrachloromercurate (TCM) to which was added a small amount of etheylenediaminetetraacetic acid disodium salt (EDTA). A dichlorosulfitomercurate complex was formed which was resistant to atmospheric oxidation by O_2, O_3 and NO_2. The EDTA eliminated any analytical interferences from adsorbed heavy metals. Prior to analyses, all solutions were stored in a refrigerator.

Analyses:

Before analyses, the impinger volume was brought back to 50 ml. using TCM solution. An aliquot (5 ml.) was then pipetted into a volumetric flask and the volume was brought up to 10 ml. with more TCM, this

mixture was allowed to stand for twenty minutes so that absorbed ozone could decompose.

To the volumetric flask was added 0.6% sulfamic acid (1 ml.) and this was allowed to stand for 10 minutes to destroy any absorbed oxides of nitrogen. Next, 0.2% of formaldehyde (2 ml.) was pipetted immediately followed by pararosaniline dye (5 ml.). It was then brought up to the 25 ml. mark with distilled water, mixed thoroughly and allowed to stand for thirty minutes.

A reagent blank and a dilute solution of sodium sulfite of known concentration were treated in the same way as above. The absorbance was determined at 548 nm. using 1 cm. optical path length cells in a spectrophotometer (Spectronic 20, Bausch and Lomb). Distilled water was used as the reference.

$$\text{Concentration of } SO_2 (ug/m^3.) = \frac{A\text{-}Ao \times 10^3 \times B}{V}$$

Where A = Sample Absorbance

Ao = reagent blank absorbance

10^3 = conversion of liters to m^3.

V = volume of air sampled in liters

B = calibration factor derived from calibration curve

Calibration:

A calibration curve was prepared using six different concentrations of sodium sulfite, the diluent being TCM. The slope of this curve gave the calibration

factor B. A calibration check at one point on the curve was conducted at each analysis session.

The spectrophotometer was calibrated once every six months using cobalt chloride (22 g.) in 1% hydrochloric acid (1 liter). The meter was set at 510 um. and the absorbance of the stock solution determined. The stock was then diluted two times and the absorbance noted. No meter adjustment was required, based on this calibration procedure. New calibration curves were prepared once every three months. A precision flow meter was used to check air flow before and after every sample period under actual field conditions. The flow meter was calibrated against a soap bubble meter.

A dynamic calibration system was conducted using a permeation tube method (Monitor Labs Inc.). The diluted gas concentrations were sampled for 24-hour periods. The amount of SO_2 permeating was determined by a weight loss method using an electrobalance (Cahn).

Many researchers have tested the sensitivity and accuracy of this manual method of monitoring SO_2. Blacker (1973) compared the bubbling method with the conductivity method using H_2O_2. The result indicated that there was a significant correlation between both methods. Two Dutch and two German Institutes conducted correlation studies based on the manual bubbling method (Lahmann, 1976). They found from the laboratory and field studies that there was very good agreement between samplers. Logsdon (1975) compared the manual method with two automated methods based on the same principle of reactions with pararosaniline dye and found that there were no differences between methods.

b. Nitrogen Dioxide (Jacobs and Hochheiser, 1958; Environmental Protection Agency, 1971).

Collection:

NO_2 collection system was the same as for SO_2 except that the bubbling solution in the impinger was 0.1M sodium hydroxide solution (50 ml.) A stable solution of sodium nitrite was formed. The flow rate to this impinger was also 0.2. l.p.m.

Analyses:

The impinger level was brought back to 50 ml. and then an aliquot (10 ml.) was transfered to a test tube. To this was added 0.03% hydrogen peroxide (1 ml.), 2% sulfanilamide solution in phosphoric acid (10 ml.) and 0.1% N-(1-naphthyl)-ethylenediamine dihydrochloride (NEDA) solution (1.4 ml.). This mixture was shaken thoroughly and allowed to stand for 10 minutes. A blank of sodium hydroxide was treated the same way as above. The absorbance was determined at 540 nm. in a spectrophotometer (Bausch and Lomb) using 1 cm. cells.

$$\text{Concentration of } NO_2 (\text{ ug./m}^3.) = \frac{\text{ug}NO_2/\text{ml.} \times 50}{V \times 0.35}$$

$\text{ug}NO_2$/ml. proportional to absorbance from calibration curve

50 = volume of reagent used in impinger (ml.)

V = volume of air sampled m^3.

0.35 = efficiency factor for method

Calibration:

A calibration curve was prepared using five known concentrations of sodium nitrite in solution. This calibration was repeated once every three months, and at every analysis session, one calibration sample was done.

The flow through the filtered impinger was checked as for SO_2.

A dynamic gas calibration was conducted using NO_2 permeation tubes in a manner similar to that described for SO_2. The absorption solution was NaOH.

The Jacobs-Hochheiser method for NO_2 was criticized by many chemists as being inefficient and variable. An EPA (1973) study showed that the method under-estimated the NO_2 levels at concentrations around 130 ug/m^3. and over-estimated the levels at concentrations below 130 ug/m^3. The writer did not change the method of monitoring since no new reference method was provided at the time and no attempts were made to correct the values since no correlation studies were available. One other area of contention was the use of fritted impingers for bubbling through NaOH. The EPA (1971) recommended the use of frits, and Purdue (1972) supported its use after comparing it with the restrictive type of impinger. In this study, frits were always used for NO_2 sampling and calibration.

APPENDIX 3.9

OUTDOOR AIR POLLUTION MEASUREMENTS

Outdoor air pollution was measured in the three towns to assess the air pollution exposure of the residents. In each town, an average exposure was obtained by using up to 5 sampling sites.

i. Sampling Sites

Ansonia:

(Fig. A.3.9.1). Ward 4, had a surface area of 2.5 km^2 (1 m^2). It was on the Western slope of the valley and most of the buildings were residential; a few were commercial. Sampling sites were located at the bottom of the valley (#C); at the 40m (125 ft) elevation level (#B); and at the 70m (225 ft) elevation, (#A). A fourth site (#D) was also located at the 40m elevation because this level had the greatest population density.

Lebanon:

(Fig. A.3.9.2). Sampling sites 1, 2, 3, 4 and 5 were positioned roughly at each of the four corners of the town and one at the geographical center. The sites were located only in populated areas since this town was sparsely populated.

Winnsboro:

(Fig. A.9.3.3). Because fewer samplers were available to monitor this town, only three sites, H, T and P were selected; one each at the two corners (H and P), (the other two corners were thinly populated) and one at the downtown center(P).

All samplers were electrically powered and manually started. They were set up on private property for security reasons. This also allowed for a source of electrical power and the use of a resident to note any mishaps. The sampler housings were at least 16m (50 ft) away from the nearest road and 11 m (35 ft) from the nearest physical obstruction. The sampling probes were about 1.4m (4 ft) above ground level and they were kept on grassy lawns to minimize the contribution of resuspended soil from the surroundings.

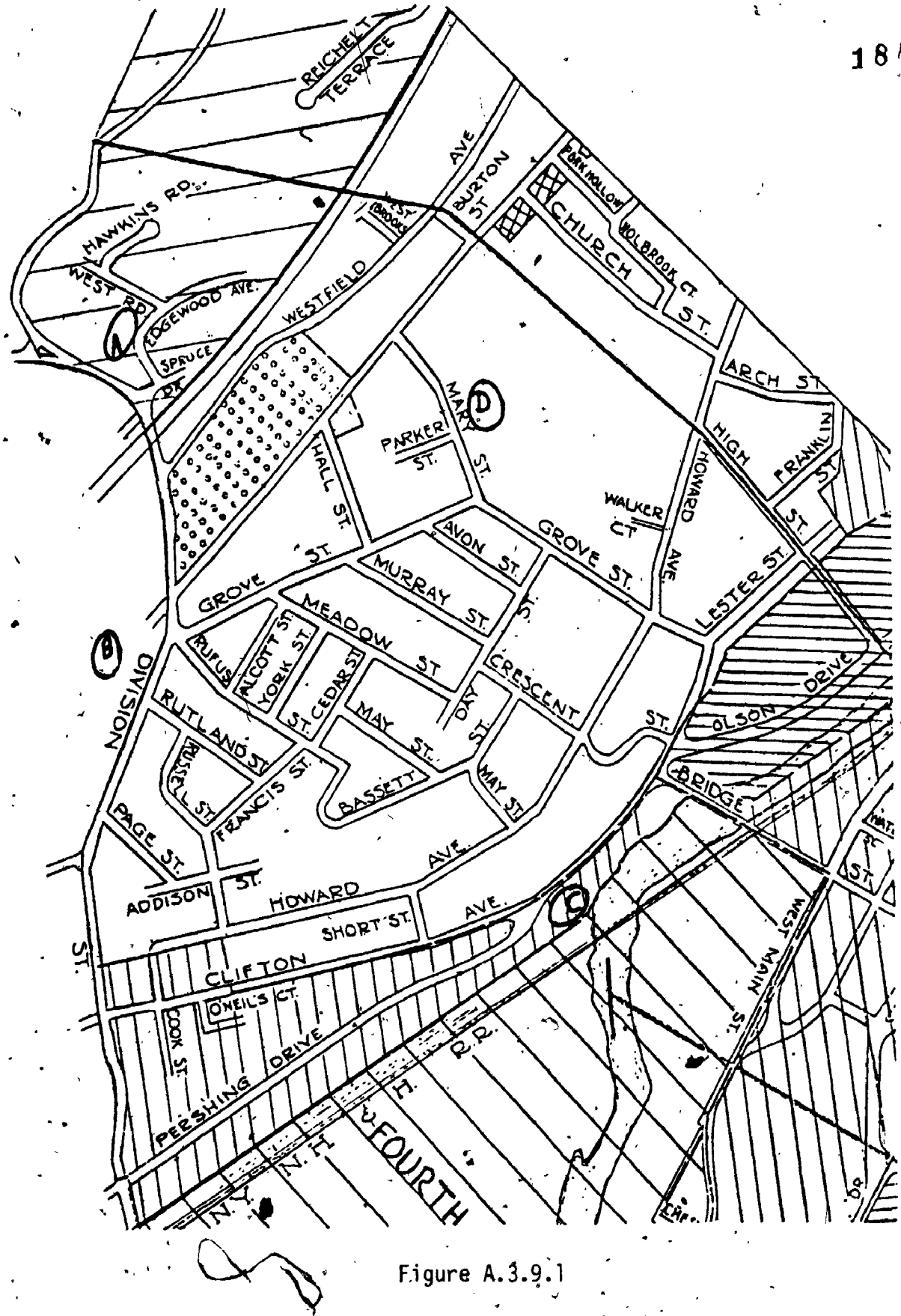


Figure A.3.9.1

Map of Ansonia, Connecticut

Showing Air Pollution Sampling Sites A, B, C and D

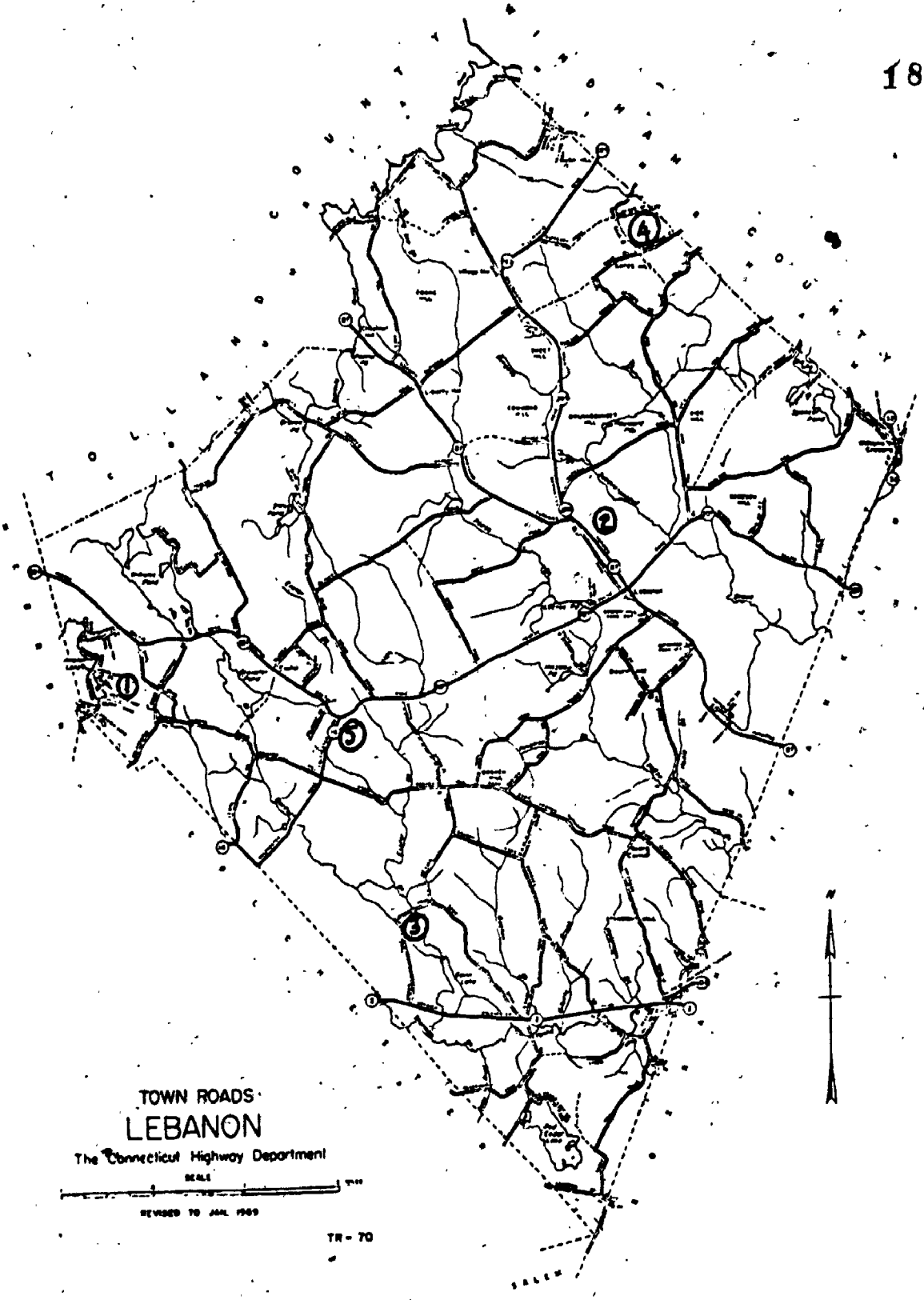


Figure A.3.9.2

Map of Lebanon, Connecticut

Showing Air Pollution Sites 1, 2, 3, 4 and 5

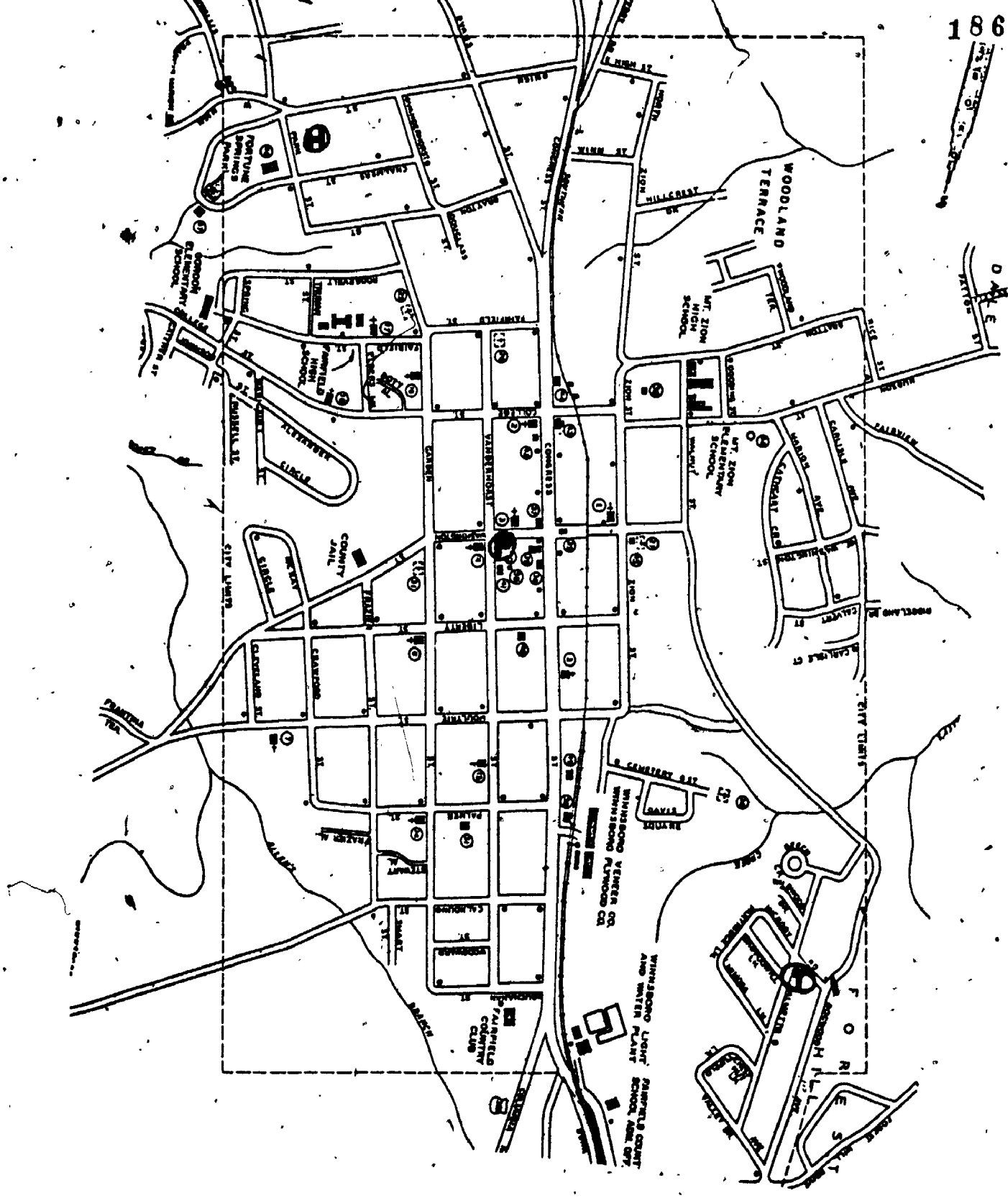


Figure A.3.9.3

Map of Winnsboro, South Carolina

Showing Air Pollution Sampling Sites H, T and P

ii. Sampling Strategy:

Most measurements in Connecticut were made on the same days in Lebanon and Ansonia, with a time difference of about 90 minutes (the time required to drive from one town to the other). Samples of all pollutants were taken over 24-hour periods so that all days of the week would be included in the sampling schedule. Every sixth day was chosen during the periods from November 16, 1972 to May 23, 1973 (Lebanon) and from January 4, 1973 to December 20, 1973 (Ansonia). From June 4, 1973 to December 20, 1973, sampling was done every twelfth day in Lebanon. From January 1, 1974 to September 30, 1974, additional samples were taken at single sites in Lebanon and Ansonia at three week intervals. During periods of domestic sampling (see Section 3.5) outdoor sampling was also conducted and these days differed from the scheduled sampling mentioned above.

In Winnsboro, South Carolina, the sampling was done on eleven consecutive days in each of the four seasons, between October 1973 and May 1975.

iii. Sample Collection Analyses and Calibration:

Each sampling site had: (1) one five-train bubbling sampler and motor (Research Appliance Cooperation) installed in a housing; and (2) a hi-volume sampler (General Metal Company) installed in another housing.

a) Sulfur dioxide was collected by absorption into potassium tetrachloromercurate solution. Chemical calibration was undertaken by using sodium sulfite. Dynamic calibration of the system was undertaken using a permeation tube method (See Appendix 3.8, for details on methods).

b) Nitrogen dioxide was collected by absorption into sodium hyroxide solution. Chemical calibration was undertaken using sodium nitrite and dynamic calibration was by the use of permeation tubes (See Appendix 3.8).

c) Total suspended particulate (TSP) was assessed by a filtration method using a hi-volume sampler and fiber glass filters. The glass fiber filter (Type A) was conditioned at 25°C and at a relative humidity of 50% for 24 hours, weighed, then loaded into a cassette. This cassette was then installed horizontally onto the hi-volume sampler. Air was drawn through the filter at a constant flow rate (40 ft^3./min.) for 24 hours; this method allowed collection of all airborne particles of size range 0.1 to 100 um. Air flow was checked five minutes after starting the sampler and at the end of the 24-hour period. The cassette was removed to the laboratory, the filter extracted, and conditioned and weighed as mentioned above.

The flow meter was calibrated once every three months using a hi-volume calibrator (General Metal Co.) with five resistance plates. The static pressure drop in the orifice, measured by a water manometer is proportional to the velocity of air through the sampler.

d) Respirable Suspended Particulates (RSP) was measured by the use of a cascade impactor (BGI Inc) fitted to the hi-volume sampler. The impactor had five stages, the collection plate on the first four stages being 2" x 5" glass fiber filter and the final stage was the normal hi-volume 8" x 10" glass fiber filter. All filters were conditioned as was mentioned earlier. The size breakdown at each stage was: stage I - above 9.4 um.; stage II - 3.8 to 9.4 um.; stage III - 2.5 to 3.8 um.; stage IV - 1.8 to 2.5 um.; stage V - 0.01 to 1.8 um.

The impactor was systematically installed at one site in each town. Because of its poor performance during wet weather, tests were not conducted on rain or snow days. In order to test what proportion of the particulate matter was less than 9.4 um., parallel sampling was conducted at one site for a number of days using two hi-volume samplers; one with the impactor and one without. To determine the mass median diameter and standard deviation, a cumulative plot was made using 2-cycle log-probability paper.

APPENDIX 4.1

Univariate Analyses on Domestic Factors, Symptoms and Lung Function.

i. Effects of Domestic Factors on Children

a. Pets (Table A.4.1)

There were no consistent trends in symptom prevalence rates between residents who had pets and those without. White male children who had pets had more cough and phlegm (RR=2.3), but this was not the case for white females. Black females who had pets reported more cough (RR=2.4) and phlegm (RR=4.1). None of these findings were significant using chi-square tests.

White children who had pets showed no better or worse lung function than those without pets except for white males who had significantly lower $MF_{25\%}$ ($P<.05$). Black children who had pets had significantly better FEV_1 ($P<.01$) than those without pets (Table A.4.1).

In general, the effects of having pets in the home were minimal on symptom reporting and lung function.

b. Hobbies (Table A.4.2)

TABLE A.4.1 EFFECT OF PETS ON SYMPTOMS AND LUNG FUNCTION IN NON-SMOKING CHILDREN

GROUP	FACTOR[≠]	NO.	SYMPTOMS (%)				RESIDUAL LUNG FUNCTION		
			Cough	Phlegm	Wheeze	Dyspnea	rMF_{25}	rMF_{50}	$rFEV_1$
White Males	w/o	117	0	.85	17.09	4.27	.10	.02	.20
	w	612	1.63	1.96	16.18	3.59	- .02*	- .01	.21
Females	w/o	98	2.04	1.02	12.24	6.12	.01	- .02	.23
	w	530	1.70	.94	12.83	5.47	- .01	- .01	.22
Black Males	w/o	203	2.46	2.96	8.37	4.43	.01	.02	.26
	w	359	2.79	1.11	11.98	5.85	.04	.06	.36***
Females	w/o	202	1.98	.50	12.87	8.91	0	.01	.32
	w	292	4.79	2.05	8.22	8.56	0	.02	.39**

≠ w/o = homes without pets

w = homes with pets

* = $p < .05$

** = $p < .01$

*** = $p < .001$

Female children who had hobbies which exposed them to gases, vapours or dusts had more symptoms in general when compared with those who had no such hobbies. White girls who had hobbies reported more phlegm (RR=9.0), $P<.001$), more wheeze (RR=1.7) and more dyspnea (RR=2.6, $P<.01$); the lung function values for this group was also lower than those without hobbies, but none of the differences were significant. Black girls who had hobbies reported more cough (RR=13.9, $P<.001$), more phlegm (RR=11.6, $P<.01$) more wheeze (RR=1.4, $P<.05$) and more dyspnea (RR=5.2, $P<.01$); these girls also had lower but not statistically significant $MF_{25\%}$ and $MF_{50\%}$. The trends for boys were inconsistent for symptom reporting and lung function.

c. Use of Fireplace (Table A.4.3)

The use of fireplaces had a variable effect on symptom reporting and lung function. In white children, those with fireplaces had slightly more symptoms (RR=1.3 to 3.1), and slightly lower FEV_1. Black boys and girls who had fireplaces showed better FEV_1, but the symptom reporting by these groups was variable.

The lack of consistent trends may indicate that the use of fireplaces may not be of any significant consequence in children's respiratory health.

TABLE A.4.2 EFFECT OF HOBBIES ON SYMPTOMS AND LUNG FUNCTION IN NON-SMOKING CHILDREN

GROUP	FACTOR≠	NO.	SYMPTOMS (%)				RESIDUAL LUNG FUNCTION		
			Cough	Phlegm	Wheeze	Dyspnea	rMF_{25}	rMF_{50}	$rFEV_1$
White Males	w/o	543	1.29	1.84	16.02	3.68	-.01	-.04*	.22
	w	186	1.61	1.61	17.20	3.76	.04	.08	.16*
Females	w/o	565	1.77	.53	11.86	4.78	0	-.01	.23
	w	63	1.59	4.76***	20.63	12.70**	-.06	-.04	.11
Black Males	w/o	515	2.91	1.94	10.27	5.43	.03	.05	.33
	w	46	0	0	15.22	4.35	0	.01	.31
Females	w/o	487	3.08	1.23	10.06	8.21	.01	.02	.36
	w	7	42.86***	14.27**	14.29*	42.86**	-.25	-.17	.39

≠ w/o = homes without hobbies

w = homes with hobbies

* = $p < .05$

** = $p < .01$

*** = $p < .001$

TABLE A.4.3 EFFECT OF FIREPLACE ON SYMPTOMS AND LUNG FUNCTION IN NON-SMOKING CHILDREN

GROUP	FACTOR≠	NO.	SYMPTOMS (%)				RESIDUAL LUNG FUNCTION		
			Cough	Phlegm	Wheeze	Dyspnea	rMF_{25}	rMF_{50}	$rFEV_1$
White Males	w/o	475	1.26	1.47/	14.53	3.37	.02	- .02	.23
	w	254	1.57	2.36	19.69*	4.37	- .03	.02	.17*
Females	w/o	395	1.52	.51	11.90	5.82	.02	0	.23
	w	233	2.15	1.72	14.16	5.15	- .04	- .05	.20
Black Males	w/o	473	2.11	2.11	10.78	4.86	.03	.06	.31
	w	89	5.62	0	10.11	7.87	- .01	- .05	.37*
Females	w/o	405	3.95	1.73	9.38	8.40	.01	.02	.35
	w	89	2.25	0	13.48	10.11	- .01	- .03	.42**

≠ w/o = homes without fireplaces

w = homes with fireplaces

* = $p < .05$

** = $p < .01$

*** = $p < .001$

d. Use of Humidifiers (Table A.4.4)

Symptoms were slightly more frequent among those who used humidifers compared with those who did not. White males and females and black males who used humidifiers reported more cough (RR=1.4 to 3.4). All children who had humidifers reported more wheeze (RR=1.1 to 1.5). There were no statistically significant effects of humidifier usage on lung function in the group of children. In general children who had a humidifier in their homes had slightly lower $MF_{25\%}$ and $MF_{50\%}$. The finding for humidifier usage is difficult to interpret since it may represent either a therapeutic usage or a cause/effect relationship.

e. Use of Air Conditioners (Table A.4.5)

Children residing in homes with air conditioning reported slightly more cough (RR=1.2 to 3.4) and wheeze (RR=1.1 to 1.6); for white females the difference for wheeze was significant ($P<.01$). White males and females and black males who lived in homes with air conditioning showed significantly better FEV_1, ($P<.001$ for white males and females, $P<.01$ for black male), but there were no consistent or significant trends with the other lung function parameters. The contradictory findings for symptoms and FEV_1 in white girls is difficult to explain.

f. Type of Domestic Heating (Table A.4.6)

Children who lived in homes with hot water and electric heating reported slightly more phlegm, wheeze and dyspnea. These children also showed

TABLE A.4.4 EFFECT OF HUMIDIFIER ON SYMPTOMS AND LUNG FUNCTION IN NON-SMOKING CHILDREN

GROUP	FACTOR≠	NO.	SYMPTOMS (%)				RESIDUAL LUNG FUNCTION		
			Cough	Phlegm	Wheeze	Dyspnea	rMF_{25}	rMF_{50}	$rFEV_1$
White Males	w/o	617	1.30	1.94	15.24	3.73	.01	0	.21
	w	112	1.79	.89	22.32	3.57	- .04	- .07	.20
Females	w/o	538	1.30	1.12	12.64	5.58	0	- .02	.21
	w	90	4.40*	0	13.37	5.56	- .03	0	.23
Black Males	w/o	540	2.59	1.85	10.56	5.37	.03	.05	.32
	w	22	4.55	0	13.64	4.55	- .06	- .13	.41
Females	w/o	465	3.66	1.29	9.89	8.60	.01	.03	.36
	w	29	3.45	3.45	13.79	10.34	- .11	- .23	.38

≠ w/o = homes without humidifiers

w = homes with humidifiers

* = $p < .05$

** = $p < .01$

*** = $p < .001$

TABLE A.4.5 EFFECT OF AIR CONDITIONING ON SYMPTOMS AND LUNG FUNCTION IN NON-SMOKING CHILDREN

GROUP	FACTOR≠	NO.	SYMPTOMS (%)				RESIDUAL LUNG FUNCTION		
			Cough	Phlegm	Wheeze	Dyspnea	rMF_{25}	rMF_{50}	$rFEV_1$
White Males	w/o	428	1.17	1.64	14.72	3.50	- .01	- .01	.12
	w	301	1.66	1.99	18.60	3.99	.03	0	.33***
Females	w/o	399	1.00	1.50	10.53	4.01	.01	.01	.13
	w	229	3.06	0	16.59**	8.30	- .04	- .06	.38***
Black Males	w/o	457	2.41	1.97	10.50	5.69	.03	.04	.31
	w	105	3.81	.95	11.43	4.81	.01	.07	.38**
Females	w/o	401	3.49	1.50	9.23	8.98	.01	.01	.37
	w	93	4.30	1.08	13.98	7.53	- .02	.02	.32

≠ w/o = homes without air conditioning

w = homes with air conditioning

* = $p < .05$

** = $p < .01$

*** = $p < .001$

lower FEV_1, the mean FEV_1 for those who lived in homes with hot water heating being consistently the lowest among all groups ($P < .001$). These trends in the FEV_1 were not seen in the other lung function parameters.

In general, homes with hot water heating may be older and probably dustier.

g. Effect of Cooking Fuel (Table A,4.7)

White boys and girls who lived in homes where gas stoves were used did not consistently report more symptoms than those who lived in homes where electric stoves were used. In some cases the relative risk was higher with those who lived in homes with electric stoves (RR=2.0 for phlegm, 1.4 for wheeze and 2.0 for dyspnea in white boys).

The FEV_1 in both white boys and girls who lived in homes with gas stoves were consistently lower ($P < .001$ for boys, $P < .01$ for girls), but these trends were not seen for the other lung function parameters.

In black boys and girls, gas stove usage was associated with better FEV_1 ($P < .01$), and slightly less cough, phlegm and wheeze in black boys. Black girls who lived in homes with gas stove had more cough (RR=3.3, $P < .01$). I am unable to explain why there are differences between whites and blacks for symptoms and FEV_1.

TABLE A.4.6 EFFECT OF HEATING ON SYMPTOMS AND LUNG FUNCTION IN NON-SMOKING CHILDREN

GROUP	FACTOR*	NO.	SYMPTOMS (%)				RESIDUAL LUNG FUNCTION		
			Cough	Phlegm	Wheeze	Dyspnea	rMF_{25}	rMF_{50}	$rFEV_1$
White Males	FA	341	1.76	1.47	14.66	2.35	- .02	- .07*	.29***
	HW	210	1.43	2.86	16.67	3.37	- .02	.08	.02
	EL	78	1.28	2.56	21.79	5.37	- .04	- .07	.24
	OT	100	0	0	17.00	8.00	.05	.08	.27
Females	FA	290	1.72	.34	12.41	5.52	0	- .01	.31***
	HW	174	1.15	1.72	12.64	5.17	0	0	.04
	EL	81	1.23	1.23	12.05	4.94	- .07	- .06	.22
	OT	83	3.61	1.20	10.84	7.23	.03	- .03	.28
Black Males	FA	211	2.84	2.84	8.06***	4.27	.06	.10	.37***
	HW	35	2.86	5.71	20.00	5.71	- .01	0	.06
	EL	64	4.69	0	17.19	3.12	.03	.01	.24
	OT	252	1.98	.79	9.92	6.75	0	.01	.34
Females	FA	173	5.20	.58	9.25	8.67	.06	.09	.41***
	HW	30	0	3.33	6.67	16.67	- .09	- .03	.06
	EL	80	5.00	1.25	13.75	5.00	- .06	- .09	.31
	OT	211	2.37	1.90	9.95	9.00	0	0	.39

* FA = Forced Air
HW = Hot Water radiators
EL = Electric heaters
OT = Other heating systems

* = $p < .05$
** = $p < .01$
*** = $p < .001$

TABLE A.4.7 EFFECT OF COOKING FUEL ON SYMPTOMS AND LUNG FUNCTION IN NON-SMOKING CHILDREN

GROUP	FACTOR≠	NO.	SYMPTOMS (%)				RESIDUAL LUNG FUNCTION		
			Cough	Phlegm	Wheeze	Dyspnea	rMF_{25}	rMF_{50}	$rFEV_1$
White Males	EL	461	2.17	2.17	18.66	4.56	- .01	- .02	.27
	GAS	268	0	1.12	12.31	2.24	.03	.01	.10***
Females	EL	411	1.70	.97	14.36	5.11	- .02	- .03	.24
	GAS	217	1.84	.92	9.68	6.45	.02	.01	.16**
Black Males	EL	232	3.01	2.11	10.84	5.12	.04	.04	.30
	GAS	230	2.17	1.30	10.43	5.65	.01	.05	.36**
Females	EL	278	1.80	1.08	10.43	8.63	- .01	- .01	.33
	GAS	216	6.02**	1.85	9.72	9.80	.02	.05	.40**

≠ EL = homes with electric stoves
GAS = homes with gas stoves

* = $p < .05$
** = $p < .01$
*** = $p < .001$

h. Domestic Crowding (Table A.4.8)

There were no consistent effects of domestic crowding on symptom prevalence rates in all the groups of children. In white children, there was a negative correlation between r FEV_1 and crowding ($r = -.17$, $P < .001$ for white males). In black children the reverse trends were seen ($r = .13$, $P < .001$ for males; $r = .19$, $P < .001$ for females).

The analysis of variance of FEV_1 showed that in white males and black males and females the differences between the crowding groups were significant ($P < .001$ for white males and black females, $P < .01$ for black males). In general, the other lung function parameters showed no consistent trends.

The inconsistent findings may be due to the small sample sizes in certain crowding groups. If the population was divided into two crowding groups i.e. ≤ 4 and ≥ 5, the FEV_1 was found to be lower in white boys and girls ($P < .05$), but not for black boys and girls in the crowded homes.

i. Number of Smokers in the Home (Table A.4.9)

Children living in homes with one smoker reported slightly more cough than children living in homes with no smokers (RR=1.5 to 2.7), but in general children living in homes with 2 or more smokers did not report more cough than those living in homes with only one smoker. The frequency of reporting phlegm was not consistent with the number of

TABLE A.4.8 EFFECT OF CROWDING ON SYMPTOMS, AND LUNG FUNCTION IN NON-SMOKING CHILDREN

GROUP	FACTOR*	NO.	SYMPTOMS (%)				RESIDUAL LUNG FUNCTION			
			Cough	Phlegm	Wheeze	Dyspnea	rMF_{25}	rMF_{50}	$rFEV_1$	
White Males	1.5	12	0	0	16.67	0	- .02	- .05	.34***	1
	3.5	219	.91	1.83	16.44	3.65	.03	.01	.25	
	5.5	343	1.75	2.33	17.49	3.75	- .03	- .02	.21	
	6.5	155	1.29	.65	13.55	3.87	.04	- .01	.11	
Females	1.5	7	0	0	0	0	- .03	- .18	.25	
	3.5	194	1.03	.52	13.40	7.22	- .01	- .04	.22	
	5.5	304	1.97	1.32	13.82	4.61	- .02	- .01	.24	
	6.5	123	2.44	.81	9.76	5.69	.05	.03	.16	
Black Males	1.5	7	0	14.29	0	0	.20	.22	.32**	2
	3.5	92	3.26	2.17	16.30	4.35	- .01	- .04	.28	
	5.5	155	1.94	1.94	10.32	5.16	0	.01	.29	
	6.5	308	2.92	1.30	9.42	5.84	.05	.09	.35	
Females	1.5	5	20.00	0	20.00	0	.14	.48	.29***	3
	3.5	70	0	0	14.29	11.43	- .14	- .11	.27	
	5.5	135	5.19	2.22	12.59	8.15	- .01	.06	.36	
	6.5	284	3.52	1.41	7.75	8.45	.05	.02	.39	

* 1.5 = Average for homes with 2 or less persons
3.5 = Average for homes with 3 and 4 persons
5.5 = Average for homes with 5 and 6 persons
6.5 = Average for homes with more than 6 persons

1 correlation coeff, r = -.17, p<.001
2 correlation coeff, r = .13, p<.001
3 correlation coeff, r = -.19, p<.001

* = p<.05
** = p<.01
*** = p<.001

smokers in the home. White children living in homes with one smoker had more wheeze than those living in homes with no smokers and 2 or more smokers; in black children the trends were inconsistent. Except for white boys, the presence of one smoker in the home was associated with slightly more reporting of dyspnea, and with black girls, the differences between the smoking groups were significant ($P < .05$).

There were no systematic differences in lung function between children living in homes with either no smokers, one smoker or 2 or more smokers.

In general, the presence of smokers in the home did not seem to have a consistent effect on the frequency of symptom reporting and lung function changes in non-smoking children.

ii. Effects of Domestic Factors on Adults

The adult black population was under-represented for males, and as a consequence, little emphasis was placed on the findings for this group.

a. Pets (Table A.4.10)

The presence of domestic pets was not associated with any effects on symptom frequency except that, in general, white adults who lived in homes without pets had slightly more symptoms; there were no consistent trends in the lung function parameters except that the mean FEV_1 of those who lived in homes with pets was slightly (but not significantly) better than those who had pets.

TABLE A.4.9 EFFECT OF NO. OF SMOKERS ON SYMPTOMS AND LUNG FUNCTION IN NON-SMOKING CHILDREN

GROUP	FACTOR≠	NO.	SYMPTOMS (%)				RESIDUAL LUNG FUNCTION		
			Cough	Phlegm	Wheeze	Dyspnea	rMF_{25}	rMF_{50}	$rFEV_1$
White Males	0	243	.41	2.06	15.23*	4.53	.03	.03	.21
	1	265	1.13	1.51	16.98	3.40	- .05	- .07	.20
	2+	221	2.71	1.81	16.74	3.17	.03	.02	.21
Females	0	195	1.54	.51	10.26	4.62	- .02	- .01	.22
	1	214	2.34	.47	15.89	6.54	- .03	- .04	.19
	2+	219	1.37	1.83	11.87	5.48	.04	.01	.24
Black Males	0	218	1.83	2.75	10.09	1.83	.02	.05	.33
	1	195	3.59	.51	9.23	7.18	.06	.08	.31
	2+	149	2.68	2.01	13.42	8.05	0	0	.33
Females	0	188	2.66	.53	10.64	5.32*	0	.07	.34
	1	165	4.85	1.21	9.70	7.88	.02	- .04	.39
	2+	141	3.55	2.84	9.91	14.18	- .01	0	.35

≠ 0 = no smokers in home
1 = one smoker in home
2+ = two or more smokers in home

* = $<.05$

There were no consistent trends in the symptom frequency or lung function in black females except those with pets reported slightly more cough (RR=1.2) and dyspnea (RR=1.7).

b. Hobbies (Table A.4.11)

White females who had hobbies which exposed them to gases, vapours and dusts reported more cough (RR=1.3), phlegm (RR=1.2), and wheeze (RR=1.3, P .05) than those who did not have such hobbies. White males with hobbies reported more phlegm (RR=1.3) and wheeze (RR=1.6). In the white adult population, there were no significant effects on the lung function parameters although generally, the residual values were higher in those subjects with hobbies. In the black female group, only four reported having hobbies which exposed them to airborne gases, vapours and dusts and hence the findings may not be representative.

c. Use of Fireplaces (Table A.4.12)

White adult males and females who had fireplaces reported less frequently on each symptom compared with those who had fireplaces; in these groups there were no consistent trends in lung function.

Black females who had fireplaces reported more cough and phlegm, but had significantly better FEV_1 ($P < .05$).

TABLE A.4.10 EFFECT OF PETS ON SYMPTOMS AND LUNG FUNCTION IN NON-SMOKING ADULTS

GROUP	FACTOR ≠	NO.	SYMPTOMS (%)				RESIDUAL LUNG FUNCTION		
			Cough	Phlegm	Wheeze	Dyspnea	rMF_{25}	rMF_{50}	$rFEV_1$
White Males	w/o	99	6.06	11.11	18.12	11.11*	0	- .14	- .07
	w	216	7.41	9.72	14.81	5.09	.06	.04	.02
Females	w/o	343	7.87	4.96	18.66	19.24	.03	- .04	- .03
	w	619	6.95	4.68	15.02	17.29	.02	- .05	.01
Black Males	w/o	42	9.52	11.90	14.29	7.14	- .04	- .33	.07
	w	17	0	11.76	23.53	5.88	.02	- .14	.04
Females	w/o	179	1.68	2.79	10.61	7.82	0	- .01	.09
	w	146	2.05	.68	11.64	13.07	- .04	- .13	.13

≠ w/o = homes without pets
w = homes with pets

* = $p < .05$
** = $p < .01$
*** = $p < .001$

TABLE A.4.11 EFFECT OF HOBBIES ON SYMPTOMS AND LUNG FUNCTION IN NON-SMOKING ADULTS

GROUP	FACTOR ≠	NO.	SYMPTOMS (%)				RESIDUAL LUNG FUNCTION		
			Cough	Phlegm	Wheeze	Dyspnea	rMF_{25}	rMF_{50}	$rFEV_1$
White Males	w/o	269	7.43	9.67	14.50	7.06	.04	- .03	- .01
	w	46	4.35	13.04	23.91	6.52	.06	.05	.03
Females	w/o	853	7.03	4.69	15.83	18.89	.02	- .06	- .01
	w	109	9.17	5.50	20.18*	10.09	.02	.06	.04
Black Males	w/o	55	7.27	12.73	18.18	7.27	- .04	- .35	.06
	w	4	0	0	0	0	.31	.64	.07
Females	w/o	321	1.87	1.87	10.28***	10.59**	- .01	- .07	.10**
	w	4	0	0	75.00	50.00	- .35	.22	.37

≠ w/o = homes without hobbies

w = homes with hobbies

* = $p < .05$

** = $p < .01$

*** = $p < .001$

TABLE A.4.12 EFFECT OF FIREPLACE ON SYMPTOMS AND LUNG FUNCTION IN NON-SMOKING ADULTS

GROUP	FACTOR ≠	NO.	SYMPTOMS (%)				RESIDUAL LUNG FUNCTION		
			Cough	Phlegm	Wheeze	Dyspnea	rMF_{25}	rMF_{50}	$rFEV_1$
White Males	w/o	203	7.39	11.33	16.26	8.37	.01	- .05	0
	w	112	6.25	8.04	15.18	4.46	.10	.05	- .02
Females	w/o	661	8.32	5.75	16.19	19.97	.02	- .06	- .01
	w	301	4.98	2.66*	16.61	13.62	.02	- .01	0
Black Males	w/o	51	7.84	11.76	15.69	7.84	- .01	- .23	.06
	w	8	0	12.50	25.00	0	- .10	- .57	.01
Females	w/o	287	1.74	1.74	12.20	11.15	- .03	- .09	.09*
	w	38	2.63	2.63	2.63	10.53	.05	.10	.20

≠ w/o = homes without fireplaces

w - homes with fireplaces

* = $p < .05$

** = $p < .01$

*** = $p < .001$

d. Use of Humidifiers (Table A.4.13)

Symptom reporting was more frequent among black and white females who lived in homes with humidifiers; for white females the difference in phlegm reporting was significant (RR=1.9, $P<.05$), and in black females the difference in wheeze reporting was significant (RR=2.8, $P<.05$). These trends in symptoms were not associated with any consistent trends in the lung function parameters between the groups.

e. Use of Air Conditioning (Table A.4.14)

The use of air conditioning was associated with slightly more phlegm (RR=1.1) wheeze (RR=1.1) and dyspnea (RR=1.7) in white males, and more phlegm (RR=1.3) and wheeze (RR=1.3) in white females. Black females who lived in homes with air conditioning reported more cough (RR=2.6), phlegm (RR=2.6), and wheeze (RR=1.3) than those who did not have air conditioning. The use of air conditioning was not associated with any consistent trends in any of the lung function parameters for white males and females. Among black residents, the lung function measurements were generally lower for those living in homes with air conditioning.

f. Type of Domestic Heating (Table A.4.15)

The use of the various types of heating systems was not associated with any consistent reporting of symptoms in any heating category in white males and females, but the lung function measurement for white males showed lower mean values for those who lived in homes with hot water

TABLE A.4.13 EFFECT OF HUMIDIFIER ON SYMPTOMS AND LUNG FUNCTION IN NON-SMOKING ADULTS

GROUP	FACTOR≠	NO.	SYMPTOMS (%)				RESIDUAL LUNG FUNCTION		
			Cough	Phlegm	Wheeze	Dyspnea	rMF_{25}	rMF_{50}	$rFEV_1$
White Males	w/o	279	6.81	10.39	16.13	7.17	.02	- .02	- .02
	w	36	8.33	8.33	13.89	5.56	.20	.17	.09
Females	w/o	815	6.99	4.17	15.09	17.91	.02	- .05	0
	w	147	8.84	8.16*	23.13	18.37	.04	.01	- .02
Black Males	w/o	55	7.27	10.91	18.18	7.27	0	- .24	.05
	w	4	0	25.00	0	0	- .27	- .79	.11
Females	w/o	311	1.61	1.61	10.29	10.61	- .01	- .06	.11
	w	14	7.14	7.14	28.57*	21.47	- .25	- .20	0

≠ w/o = homes without humidifiers

w = homes with humidifiers

* = $p < .05$

** = $p < .01$

** = $p < .001$

TABLE A-4.14 EFFECT OF AIR CONDITIONING ON SYMPTOMS AND LUNG FUNCTION IN NON-SMOKING ADULTS

GROUP	FACTOR ≠	NO.	SYMPTOMS (%)				RESIDUAL LUNG FUNCTION		
			Cough	Phlegm	Wheeze	Dyspnea	rMF_{25}	rMF_{50}	$rFEV_1$
White Males	w/o	186	7.53	9.68	15.05	5.38	.04	0	- .01
	w	129	6.20	10.85	17.05	9.30	.04	- .04	0
Females	w/o	565	7.08	4.25	14.69	18.05	.01	- .07	- .01
	w	397	7.56	5.54	18.64	17.88	.04	- .01	0
Black Males	w/o	43	6.98	11.63	20.93	9.30	- .01	- .19	.09
	w	16	6.25	12.50	6.25	0	- .04	- .51	- .03
Females	w/o	273	1.47	1.47	10.42	11.36	0	- .06	.11
	w	52	3.85	3.85	13.46	9.62	- .11	- .09	.07

≠ w/o = homes without air conditioning

w = homes with air conditioning

* = $p < .05$

** = $p < .01$

*** = $p < .001$

heating; for white females there were lower mean values for those who had hot water and electric heating; but none of these differences were significant.

In black females, those who lived in homes with hot water heating reported consistently more cough, phlegm, wheeze and dyspnea, and for all symptoms, the differences between the heating categories were significant ($P < .05$).

The mean lung function was also lowest in black females who lived in homes with hot water heating; the analysis of variance showed that the differences in the means between the heating groups were significant ($P < .05$ for r $MF_{25\%}$ and r $MF_{50\%}$, $P < .001$ for r FEV_1).

g. Type of Cooking Fuel (Table A.4.16)

White females who had gas stoves in their homes reported slightly more cough (RR=1.3), phlegm (RR=1.1), and dyspnea (RR=1.2); they had also marginally lower lung function. The opposite trends were seen for white males and for black females. In the case of black females, users of gas stoves showed significantly better $MF_{25\%}$ ($P < .05$).

h. Domestic Crowding (Table A.4.17)

In white males and females there appeared to be a consistent effect of crowding on symptom reporting and lung function in that, in general, residents who lived in homes with fewer persons had more symptoms and

A-4.15 EFFECT OF HEATING ON SYMPTOMS AND LUNG FUNCTION

IN NON-SMOKING ADULTS

GROUP	FACTOR ≠	NO.	SYMPTOMS (%)				RESIDUAL LUNG FUNCTION		
			Cough	Phlegm	Wheeze	Dyspnea	rMF_{25}	rMF_{50}	$rFEV_1$
White Males	FA	134	8.96	10.45	16.42	18.21	.05*	- .07	.01
	HW	123	5.69	8.94	16.26	15.69	- .07	- .09	- .04
	EL	27	11.11	3.70	18.52	0	.20	.18	.04
	OT	31	0	19.35	9.68	12.90	.29	.30	.01
Females	FA	471	8.07	6.37	15.50	16.99*	.03	- .02	0
	HW	307	7.17	3.58	16.61	19.87	.02	- .09	- .01
	EL	78	5.13	2.56	16.67	10.26	.02	- .08	- .05
	OT	106	5.66	2.83	18.87	22.64	.07	.01	.05
Black Males	FA	22	0 *	18.18***	9.09	4.55	.08	- .21	.08
	HW	5	40.00	60.00	40.00	20.00	- .22	- .29	- .20
	EL	5	0	0	20.00	0	- .05	- .06	.16
	OT	27	7.41	0	18.52	7.41	- .05	- .37	.07
Females	FA	101	.99**	1.98***	11.88*	9.90*	- .07*	- .05*	.12***
	HW	30	10.00	10.00	30.00	20.00	- .26	- .49	- .09
	EL	20	5.00	5.00	5.00	5.00	.03	- .02	.08
	OT	174	.57	0	8.05	10.92	.04	0	.13

≠ FA = Forced Air
HW = Hot Water radiators
EL = Electric heaters
OT = Other heating systems

* = $p < .05$
** = $p < .01$
*** = $p < .001$

TABLE A.4.16 EFFECT OF COOKING FUEL ON SYMPTOMS AND LUNG FUNCTION IN NON-SMOKING ADULTS

GROUP	FACTOR ≠	NO.	SYMPTOMS (%)				RESIDUAL LUNG FUNCTION		
			Cough	Phlegm	Wheeze	Dyspnea	rMF_{25}	rMF_{50}	$rFEV_1$
White Males	EL	198	7.58	11.62	16.67	16.57	.01	- .01	- .02
	GAS	117	5.98	9.69	14.53	17.69	.08	- .02	.02
Females	EL	596	6.54	4.53	16.44	16.44	.03	- .03	0
	GAS	366	8.47	5.19	16.12	20.49	0	- .07	- .01
Black Males	EL	31	9.68	9.68	16.13	6.45	.04	- .28	.01
	GAS	28	3.57	14.29	17.86	7.14	- .08	- .27	.10
Females	EL	175	2.86	2.86	12.00	12.57	- .09*	- .11	.09
	GAS	150	.67	.67	10.00	9.33	.06	- .01	.12

≠ EL = homes with electric stoves
GAS = homes with gas stoves

* = $p < .05$
** = $p < .01$
*** = $p < .001$

lower lung function; for white females there were significant differences in phlegm frequency between the four crowding groups such that the least crowded group had the highest reporting rate and the most crowded group had the lowest rate; for the $MF_{50\%}$ in white males, there was significant positive correlation with crowding ($P < .01$). In the black female group the trends were not as consistent for the symptom prevalence rates. The analysis of variance on the r FEV_1 showed significant differences between the means in the four crowding groups ($P < .001$), and there was a significant positive correlation between r FEV_1 and crowding ($P < 001$).

i. Number of Smokers in the home (Table A.4.18)

The presence of one or more smoker's in the home was not associated with any increased reporting of symptoms in the non-smoking residents, and in general the symptom frequency rates were marginally higher in those residents who lived in homes with no smokers. In white males there appeared to be a dose response with the r FEV_1, and in white females with the r $MF_{50\%}$, but neither of these was significant; in addition the other lung function measurements were inconsistent.

TABLE A.4.17 EFFECT OF CROWDING ON SYMPTOMS AND LUNG FUNCTION

IN NON-SMOKING ADULTS

GROUP	FACTOR ≠	NO.	SYMPTOMS (%)				RESPIRATORY LUNG FUNCTION		
			Cough	Phlegm	Wheeze	Dyspnea	rMF_{25}	rMF_{50}	$rFEV_1$
White Males	1.5	87	6.90	16.09	23.00	12.64	- .05	- .24[1]	- .05
	3.5	132	6.82	9.85	11.36	6.06	.06	- .02	0
	5.5	71	4.23	2.82	15.40	2.82	.05	.15	.03
	6.5	25	16.00	12.00	16.00	4.00	.19	.27	.02
Females	1.5	317	10.73	7.57**	20.19	18.61	.04	- .06	- .03
	3.5	396	5.81	3.03	15.66	17.42	.03	- .02	0
	5.5	203	5.91	4.43	11.33	17.24	- .02	- .11	.01
	6.5	46	2.17	2.17	17.39	21.74	- .01	.07	.01
Black Males	1.5	18	0	11.11	16.67	5.56	- .23[2]	- .33	.06
	3.5	9	11.11	33.33	11.11	11.11	- .13	- .47	.07
	5.5	17	17.65	11.76	29.41	11.76	- .06	- .30	.03
	6.5	15	0	0	6.67	0	.35	- .07	.07
Females	1.5	48	0	2.08	2.08	10.42	.04	- .04	- .02***[3]
	3.5	85	1.18	0	12.94	14.12	- .11	- .16	.11
	5.5	81	3.70	1.23	14.81	8.64	.04	- .01	.08
	6.5	111	1.80	3.60	10.81	10.81	- .02	- .04	.17

≠ 1.5 - Average for homes with 2 or less persons

3.5 - Average for homes with 3 and 4 persons

5.5 - Average for homes with 5 and 6 persons

6.5 - Average for " " more than 6 persons

* = $p < .05$

** = $p < .01$

*** = $p < .001$

[1] Sign correlation, r = .15, $p < .01$

[2] Sign correlation, r = .37, $p < .01$

[3] Sign correlation, r = .24, $p < .001$

TABLE A.4.18 EFFECT OF NO. OF SMOKERS ON SYMPTOMS AND LUNG FUNCTION

IN NON-SMOKING ADULTS

GROUP	FACTOR #	NO.	SYMPTOMS (%)				RESIDUAL LUNG FUNCTION		
			Cough	Phlegm	Wheeze	Dyspnea	rMF_{25}	rMF_{50}	$rFEV_1$
White Males	0	222	8.04	11.61	16.07	8.48	.01	- .03	.01
	1	61	6.56	8.20	14.75	4.92	.08	.06	- .03
	2+	30	0	3.33	16.67	0	.10	- .08	- .10
Females	0	540	7.78	5.00	16.11	16.85	.03	- .03	- .01
	1	303	6.93	5.61	15.18	19.47	.04	- .05	.01
	2+	119	5.88	1.68	20.17	19.33	- .03	- .10	.01
Black Males	0	45	4.44	13.33	20.00	4.44	- .08[1]	- .26	.06
	1	7	28.57*	14.29	14.29	28.57*	.08	- .43	- .07
	2+	7	0	0	0	0	.29	- .21	.19
Females	0	159	1.26	1.89	13.21	15.09	0	- .09	.08
	1	99	2.02	2.02	9.09	7.07	- .07	- .06	.14
	2+	67	2.99	1.49	8.96	7.46	.01	- .01	.11

# 0 = no smokers in home
1 = one smoker in home
2+ = two or more smokers in home

* = $p < .05$
** = $p < .01$
*** = $p < .001$

[1] Sign correlation ($r = .26$, p .05)

APPENDIX 4.2

MULTIFACTORIAL ANALYSES BETWEEN THE EXPOSURE VARIABLES, LUNG FUNCTION AND SYMPTOMS

Legend for tables that follow:

a. Exposure variables are: HB = hobbies, FP = fireplace,
AC = air condition
HT = heating,
CK = cooking fuel
CR = crowding
No.S = number of smokers

b. Subject variables are: WM = white males
WF = white females
BM = black males
BF = black females

c. Level of significance: - = not significant
x = P < .05
xx = P < .01
xxx = P < .001

d. The tables show the significant interactions between the various exposure variables.

APPENDIX 4.2.1

INTERACTION BETWEEN EXPOSURE VARIABLES FOR $rFEV_1$ IN CHILDREN AND ADULTS

ADULTS \ CHILDREN	HB	FP	HM	AC	HT	CK	CR	No.S
HB								
WM		-	-	-	-	-	-	-
WF		-	xx	-	-	-	-	-
BM		-	-	-	x	-	-	x
BF		-	-	-	-	-	-	-
FP								
WM	-		-	-	-	-	-	-
WF	-		-	-	-	-	-	-
BM	-		-	-	-	x	-	-
BF	-		-	-	-	-	-	-
HM								
WM	-	-		-	-	-	-	-
WF	-	-		-	-	-	-	-
BM	-	-		-	-	-	-	-
BF	-	-		-	-	-	-	-
AC								
WM	x	-	-		xxx	-	x	x
WF	-	-	-		xxx	x	x	x
BM	-	xx	-		-	-	-	-
BF	-	-	-		-	-	-	x
HT								
WM	-	-	-	-		xxx	-	-
WF	-	-	-	-		xx	xx	xx
BM	-	-	-	-		xxx	x	xx
BF	-	-	-	-		xx	xxx	xx
CK								
WM	-	x	-	-	x		-	-
WF	-	-	-	-	-		xx	x
BM	-	-	-	-	-		-	-
BF	-	-	-	-	-		-	-
CR								
WM	-	-	-	-	-	-		-
WF	-	-	x	-	-	-		-
BM	-	-	-	-	xxx	-		-
BF	-	-	-	-	-	-		-
No.S								
WM	-	-	-	-	-	-	-	
WF	-	-	-	-	-	-	-	
BM	-	-	-	-	-	-	-	
BF	-	x	-	-	-	x	-	

APPENDIX 4.2.2

INTERACTION BETWEEN EXPOSURE VARIABLES FOR $rMF_{25\%}$ IN CHILDREN AND ADULTS

ADULTS \ CHILDREN	HB	FP	HM	AC	HT	CK	CR	No.S
HB								
WM		-	-	-	-	-	-	xxx
WF		-	-	-	-	-	-	-
BM		-	-	-	-	-	-	-
BF		-	-	-	-	-	-	-
FP								
WM	-		xx	-	-	-	-	-
WF	-		-	-	-	-	-	-
BM	-		-	-	-	-	-	-
BF	-		-	-	-	-	-	-
HM								
WM	-	-		-	-	-	-	x
WF	-	-		-	-	-	-	-
BM	-	-		-	-	-	-	-
BF	-	-		-	-	-	-	-
AC								
WM	-	-	-		-	-	-	-
WF	-	-	-		-	-	-	-
BM	-	-	-		x	-	xx	-
BF	-	-	-		-	-	-	-
HT								
WM	-	-	-	x		-	-	-
WF	-	-	-	xx		-	-	-
BM	-	-	-	-		-	x	-
BF	-	-	-	-		-	-	-
CK								
WM	-	-	-	-	x		-	-
WF	-	-	-	-	-		-	-
BM	-	-	x	-	-		-	-
BF	-	x	-	-	xxx		-	-
CR								
WM	-	-	-	-	-	-		-
WF	x	-	-	-	-	-		-
BM	-	-	-	-	-	-		-
BF	-	-	-	-	-	-		-
No.S								
WM	x	-	-	-	-	-	-	
WF	-	-	-	-	-	-	-	
BM	-	-	-	-	-	-	-	
BF	-	-	-	-	-	x	-	

APPENDIX 4.2.3

INTERACTION BETWEEN EXPOSURE VARIABLES FOR $rMf_{50\%}$ IN CHILDREN AND ADULTS

ADULTS \ CHILDREN	HB	FP	HM	AC	HT	CK	CR	No.S
HB								
WM		x	-	-	-	-	-	xxx
WF		-	-	-	-	-	-	-
BM			-	-	-	-	-	-
BF		-	-	-	-	-	-	-
FP								
WM	-		x	-	-	-	-	-
WF	-		-	-	x	-	-	-
BM	-		-	-	-	x	-	-
BF	-		-	-	x	-	-	-
HM								
WM	-	-		-	-	-	-	-
WF	-	-		-	-	-	-	-
BM	-	-		-	-	-	-	-
BF	-	-		-	-	-	-	-
AC								
WM	-	-	-		-	-	xx	-
WF	-	-	-		-	-	-	-
BM	-	-	-		xx	-	xxx	-
BF	-	-	-		-	-	-	-
HT								
WM	-	-	-	x		-	-	-
WF	-	-	-	xx		x	-	-
BM	-	-	-	-		-	x	-
BF	-	-	-	-		-	-	-
CK								
WM	-	-	-	-	x		-	-
WF	-	-	-	-	-		-	-
BM	-	-	-	-	-		-	-
BF	-	-	-	-	xx		-	-
CR								
WM	-	-	-	-	-	-		-
WF	-	-	-	-	-	-		x
BM	-	-	-	-	-	-		-
BF	-	-	-	-	-	-		-
No.S								
WM	x	-	-	-	-	-	-	
WF	-	-	-	-	-	-	-	
BM	-	-	-	-	-	-	-	
BF	-	-	-	-	-	-	-	

APPENDIX 4.2.4

INTERACTION BETWEEN EXPOSURE VARIABLES FOR COUGH IN CHILDREN AND ADULTS

ADULTS \ CHILDREN	HB	FP	HM	AC	HT	CK	CR	No.S
HB								
WM		-	-	-	-	-	-	-
WF		-	-	-	-	-	-	-
BM		-	-	-	-	-	-	-
BF		-	-	xx	xxx	-	xxx	xx
FP								
WM	-		-	-	-	-	-	-
WF	-		-	-	-	-	-	-
BM	-		-	-	x	-	-	-
BF	-		-	-	-	-	-	-
HM								
WM	-	-		-	-	-	-	-
WF	-	-		-	-	-	-	xx
BM	-	-		-	-	x	xxx	-
BF	-	-		-	-	-	-	-
AC								
WM	-	-	-		-	-	-	-
WF	-	-	xx		-	-	x	-
BM	-	-	-		-	-	xx	-
BF	-	-	-		-	-	-	-
HT								
WM	-	-	-	-		-	-	-
WF	-	-	x	-		-	-	-
BM	-	-	-	-		-	-	xx
BF	-	-	-	-		-	xxx	-
CK								
WM	-	-	-	-	-		-	-
WF	-	-	-	-	-		-	-
BM	-	-	-	-	-		-	-
BF	-	-	-	-	-		-	-
CR								
WM	-	-	-	-	-	-		-
WF	-	-	-	-	-	-		xx
BM	-	-	-	-	xx	-		-
BF	-	-	x	-	xx	-		-
No.S								
WM	-	-	-	-	-	-	-	
WF	-	-	-	-	-	-	-	
BM	-	-	-	xxx	-	-	x	
BF	-	-	-	x	-	-	-	

APPENDIX 4.2.5

INTERACTION BETWEEN EXPOSURE VARIABLES FOR PHLEGM IN CHILDREN AND ADULTS

ADULTS \ CHILDREN	HB	FP	HM	AC	HT	CK	CR	No.S
HB								
WM		-	-	-	-	-	-	-
WF		-	-	x	x	-	-	-
BM		-	-	-	-	-	-	-
BF		-	-	-	xxx	xxx	xx	xxx
FP								
WM	-		-	-	-	-	-	-
WF	-		-	-	-	-	x	-
BM	-		-	-	-	-	-	-
BF	-		-	-	-	-	-	-
HM								
WM	-	-		-	-	-	-	-
WF	-	-		-	-	-	-	xx
BM	-	-		-	-	-	-	-
BF	-	-		-	-	-	-	-
AC								
WM	x	-	-		-	-	-	-
WF	-	-	-		-	-	-	-
BM	-	-	-		x	-	xxx	-
BF	-	x	-		-	-	-	-
HT								
WM	-	-	-	-		-	-	-
WF	-	-	-	-		xx	-	-
BM	-	-	x	-		-	xxx	-
BF	-	-	-	-		-	-	-
CK								
WM	-	-	-	-	-		-	-
WF	-	-	-	-	-		-	-
BM	-	-	-	-	xx		-	-
BF	-	-	-	-	x		-	-
CR								
WM	-	-	-	-	-	-		-
WF	-	-	-	-	-	-		-
BM	-	-	-	-	x	-		-
BF	-	-	xx	xxx	x	-		-
No.S								
WM	-	-	-	-	-	-	-	
WF	-	-	-	-	-	-	-	
BM	-	-	-	-	-	-	-	
BF	-	-	-	x	-	-	-	

APPENDIX 4.2.6

INTERACTION BETWEEN EXPOSURE VARIABLES FOR WHEEZE IN CHILDREN AND ADULTS

ADULTS \ CHILDREN	HB	FP	HM	AC	HT	CK	CR	No.S
HB								
WM		-	-	-	-	-	-	-
WF		-	-	-	-	-	-	-
BM		-	-	-	x	-	-	-
BF		-	-	-	-	-	-	-
FP								
WM	-		-	-	-	-	-	-
WF	-		-	-	-	-	-	-
BM	-		-	-	xxx	-	-	-
BF	-		-	-	-	-	-	x
HM								
WM	-	-		xxx	-	-	-	-
WF	-	-		-	-	-	-	x
BM	-	-		-	-	-	-	-
BF	-	-		-	-	x	-	-
AC								
WM	-	-	-		-	-	-	-
WF	-	-	-		-	-	-	xx
BM	-	-	-		-	-	-	-
BF	-	-	xx		-	-	-	x
HT								
WM	x	x	-	-		-	-	-
WF	-	-	x	-		-	-	-
BM	-	-	-	-		-	-	-
BF	-	-	-	-		-	x	-
CK								
WM	-	-	-	x	-		-	-
WF	-	-	-	-	-		-	-
BM	-	-	-	-	-		-	-
BF	-	-	xx	-	-		-	-
CR								
WM	x	-	-	-	-	-		-
WF	-	-	-	-	-	-		-
BM	-	-	-	-	-	-		-
BF	-	-	-	-	-	-		-
No.S								
WM	-	-	-	-	-	xx	-	
WF	-	-	-	-	-	-	-	
BM	-	-	-	-	-	-	-	
BF	x	-	xx	-	-	-	-	

APPENDIX 4.2.7

INTERACTION BETWEEN EXPOSURE VARIABLES FOR DYSPNEA IN CHILDREN AND ADULTS

ADULTS \ CHILDREN	HB	FP	HM	AC	HT	CK	CR	No.S
HB								
WM		-	-	-	-	-	-	-
WF		-	-	xx	-	-	xxx	xxx
BM		-	-	-	-	-	-	-
BF		-	-	x	x	x	-	-
FP								
WM	-		-	-	-	-	-	-
WF	-		-	-	xx	-	-	-
BM	-		-	-	-	-	-	-
BF	-		-	-	-	-	-	-
HM								
WM	-	-		-	-	-	-	-
WF	-	-		-	-	xx	-	-
BM	-	-		-	-	-	xx	-
BF	-	-		-	-	x	xx	x
AC								
WM	x	x	-		-	-	x	-
WF	-	-	-		-	-	-	-
BM	-	-	-		-	-	-	-
BF	-	-	-		-	-	-	-
HT								
WM	-	xx	-	-		-	-	-
WF	-	-	-	-		-	-	-
BM	-	-	-	-		-	-	-
BF	-	-	-	-		-	-	-
CK								
WM	-	-	-	xx	x		-	-
WF	-	-	xx	-	-		-	-
BM	-	-	-	-	-		-	-
BF	-	-	-	-	-		-	-
CR								
WM	-	-	-	x	-	-		-
WF	-	-	-	-	-	-		-
BM	-	-	-	-	-	-		-
BF	-	-	-	-	-	-		-
No.S								
WM	-	-	-	-	-	-	-	
WF	-	-	-	-	-	-	-	
BM	-	-	-	xx	x	-	xx	
BF	-	-	-	-	-	-	-	

APPENDIX 4.3 FREQUENCY BETWEEN PAIRS OF EXPOSURE VARIABLES (%)

Legend for appendices 4.3.1 to 4.3.4:

a. Exposure variables:

CK = cooking
CR = crowding
No.S = number of smokers
AC = air conditioning
HB = hobbies
HT = heating
FA = forced air
HW = hot water
EL = electric
CR1 = crowding with less than 5 persons
CR2 = crowding with 5 or more people

APPENDIX 4.3.1

FREQUENCY BETWEEN PAIRS OF EXPOSURE VARIABLES (%) FOR WHITE CHILDREN

		HT			AC	No.S			CR		HB
MALES		FA	HW	EL	YES	0	1	≥2	CR1	CR2	YES
CK											
	ELEC	62	62	91	70	66	66	56	62	61	63
	GAS	38	38	9	30	34	34	44	38	39	37
CR											
	CR1	35	36	30	35	41	33	19			
	CR2	65	64	70	65	59	67	81			
No.S											
	0	35	32	38	36						
	1	35	38	29	37						
	≥2	30	29	32	25						
AC											
	YES	61	16	11							
HB											
	YES	35	38	13	8	34	35	31	30	70	
FEMALES											
CK											
	ELEC	66	65	81	72	65	65	65	66	66	68
	GAS	34	35	18	28	35	35	35	34	34	32
CR											
	CR1	33	31	26	39	39	36	21			
	CR2	67	69	74	61	61	64	79			
No.S											
	0	33	26	33	37						
	1	34	36	28	34						
	≥2	32	38	38	28						
AC											
	YES	62	13	37							
HB											
	YES	33	43	19	3	25	33	41	27	73	

APPENDIX 4.3.2

FREQUENCY BETWEEN PAIRS OF EXPOSURE VARIALBES (%) FOR BLACK CHILDREN

		HT			AC	No.S			CR		HB
MALES		FA	HW	EL	YES	0	1	2	CR1	CR2	YES
CK											
	ELEC	53	82	72	66	58	56	64	71	59	72
	GAS	47	17	28	33	42	43	36	29	41	28
CR											
	CR1	18	26	16	20	24	20	10			
	CR2	82	74	84	80	70	80	90			
No.S											
	0	43	34	39	47						
	1	28	37	36	30						
	≥2	28	28	25	23						
AC											
	YES	39	5	17							
HB											
	YES	41	7	13	14	54	28	17	26	74	
FEMALES											
CK											
	ELEC	50	90	80	63	58	53	57	50	57	43
	GAS	50	10	20	37	42	47	43	50	43	57
CR											
	CR1	13	17	16	29	25	9	10			
	CR2	87	83	84	71	75	91	90			
No.S											
	0	33	27	39	48						
	1	38	43	36	28						
	≥2	29	30	25	24						
AC											
	YES	38	7	24							
HB											
	YES	57	0	14	14	43	43	4	43	57	

APPENDIX 4.3.3

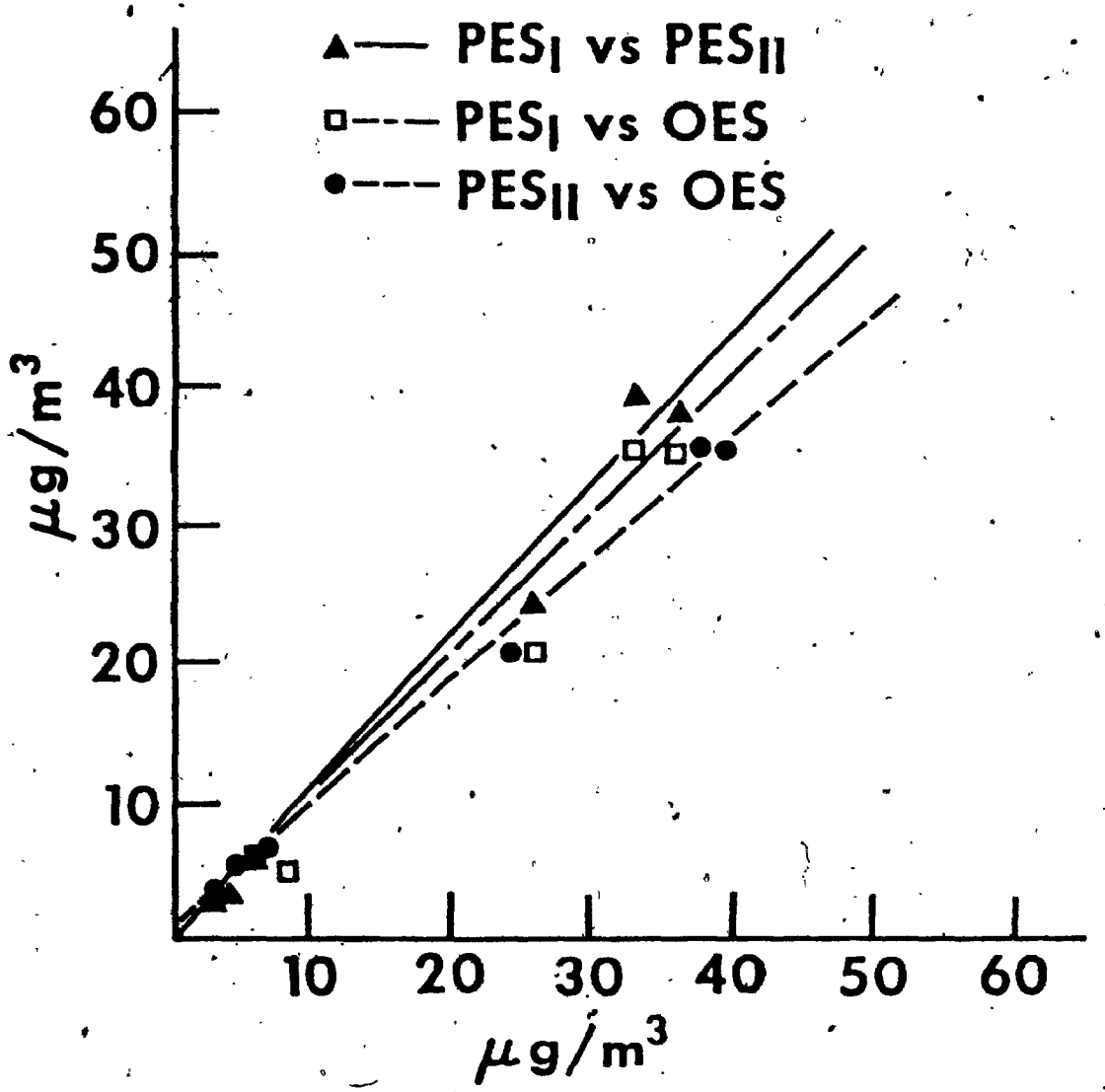
FREQUENCY BETWEEN PAIRS OF EXPOSURE VARIABLES (%) FOR WHITE ADULTS

		HT			AC	No.S			CR		HB
MALES		FA	HW	EL	YES	0	1	≥2	CR1	CR2	YES
CK											
	ELEC	64	80	80	56	49	71	57	61	50	63
	GAS	36	20	20	44	51	29	43	39	50	37
CR											
	CR1	46	80	40	50	49	57	14			
	CR2	54	20	60	50	51	43	86			
No.S											
	0	86	80	60	63						
	1	9	20	20	25						
	≥2	5	0	20	12						
AC											
	YES	44	13	19							
HB											
	YES	35	48	7	5	76	15	8	35	65	
FEMALES											
CK											
	ELEC	60	87	90	58	55	57	46	54	62	
	GAS	40	13	10	42	45	43	54	46	38	
CR											
	CR1	47	40	35	50	56	39	7			
	CR2	53	60	65	50	44	61	93			
No.S											
	0	53	37	50	63						
	1	35	37	30	25						
	≥2	12	26	20	11						
AC											
	YES	44	15	11							
HB											
	YES	44	36	12	4	51	36	13	27	73	

APPENDIX 4.3.4.

FREQUENCY BETWEEN PAIRS OF EXPOSURE VARIABLES (%) FOR BLACK ADULTS

		HT			AC	No.S			CR		HB
MALES		FA	HW	EL	YES	0	1	≥2	CR1	CR2	YES
CK											
	ELEC	53	82	72	66	58	56	64	72	59	50
	GAS	47	17	28	34	42	44	36	28	41	50
CR											
	CR1	18	26	16	20	24	16	10			
	CR2	82	74	84	80	76	84	90			
No.S											
	0	43	34	39	47						
	1	28	37	36	30						
	≥2	28	28	25	23						
AC											
	YES	39	5	17							
HB											
	YES	50	0	0	6	100	0	0	25	75	
FEMALES											
CK											
	ELEC	64	60	87	71	64	61	56	62	60	50
	GAS	36	40	13	29	30	39	44	38	40	50
CR											
	CR1	74	77	64	78	82	72	43			
	CR2	26	23	36	22	18	28	57			
No.S											
	0	59	51	47	57						
	1	28	37	38	30						
	≥2	13	12	14	13						
AC											
	YES	64	21	7							
HB											
	YES	25	0	0	0	25	50	25	0	100	

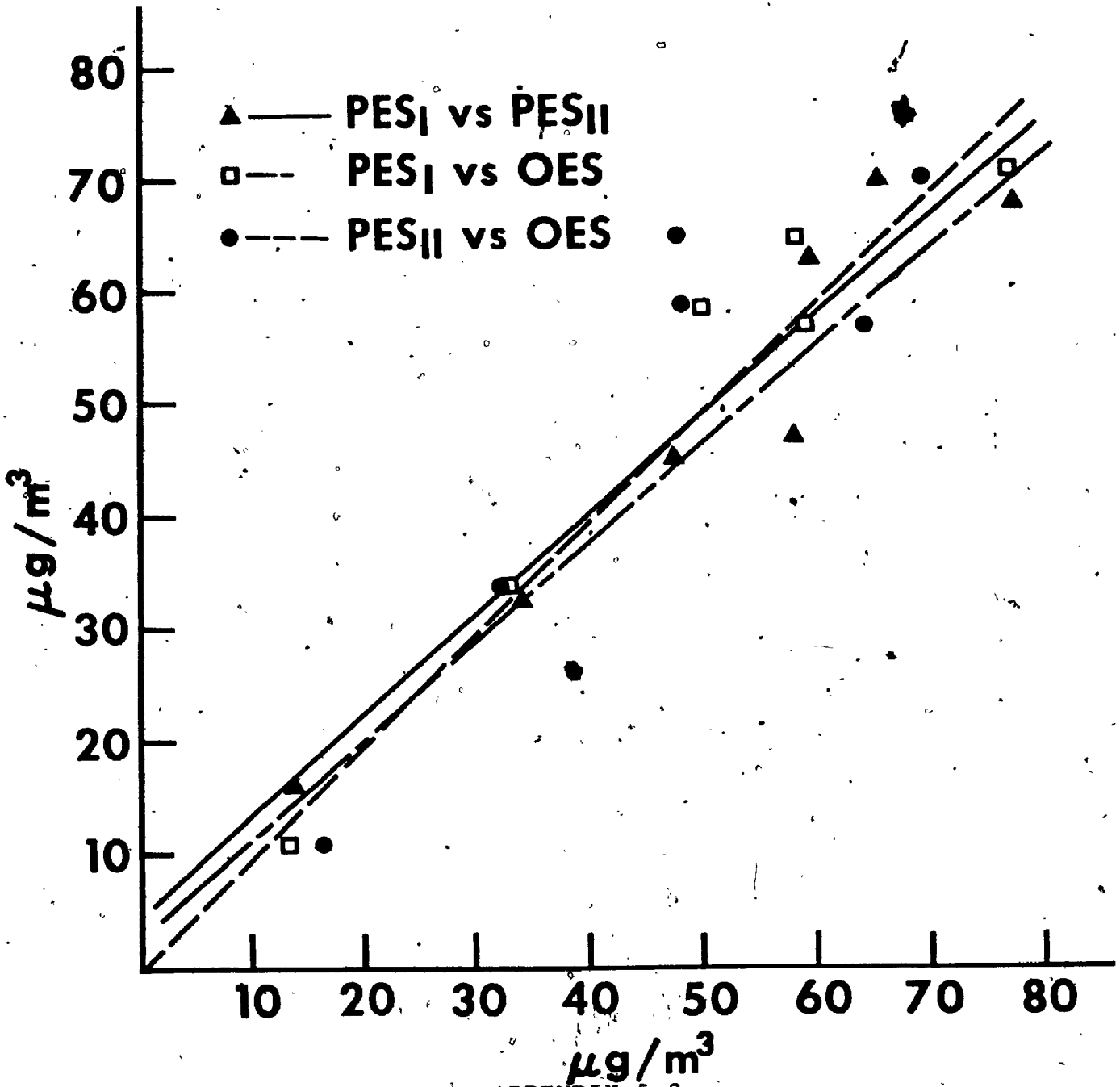


PES = Personal Environment Sampler Nos. I and II.

OES = Outside Environment Sampler

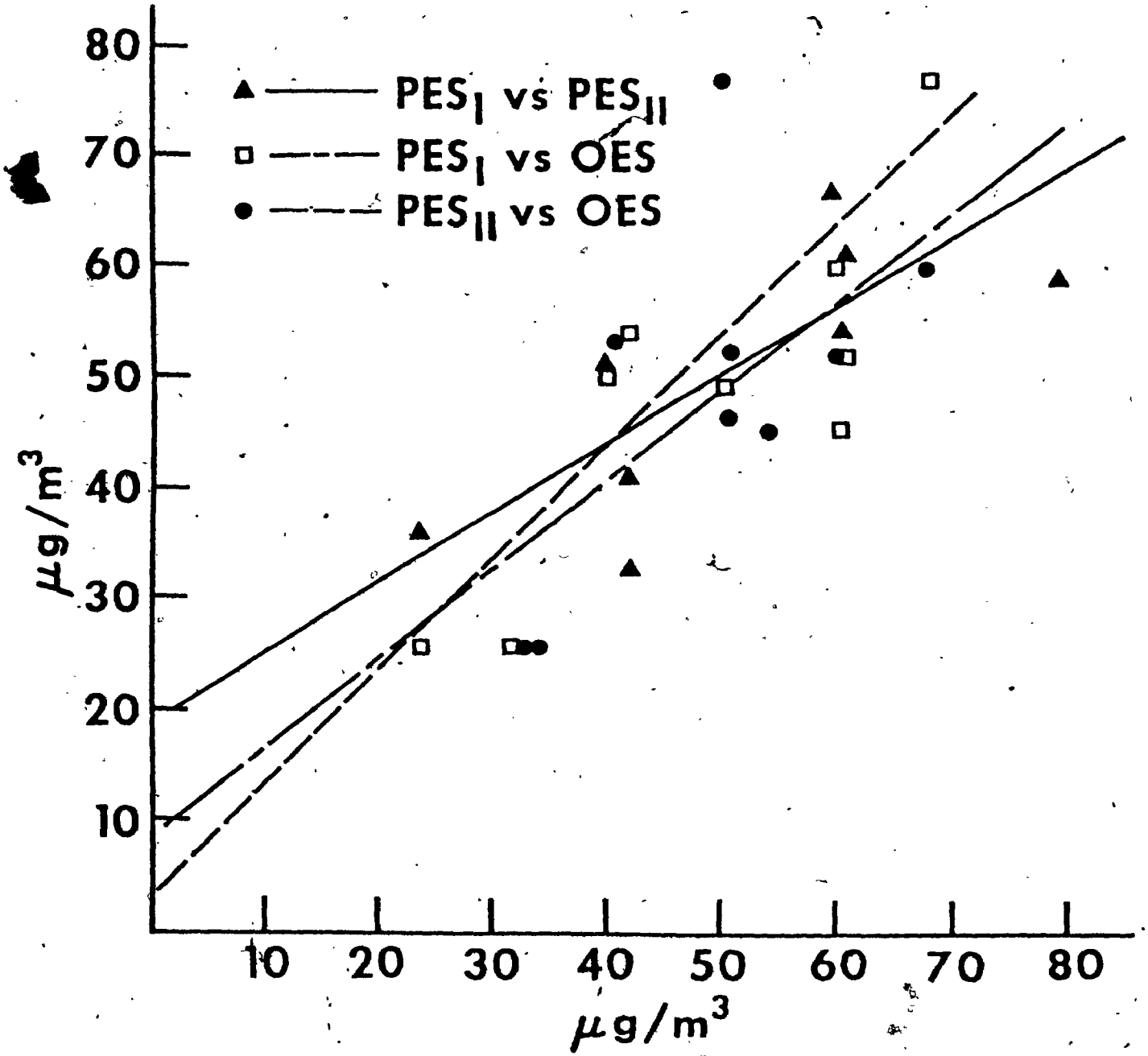
APPENDIX 5.1

Intersampler Correlation Lines for SO_2



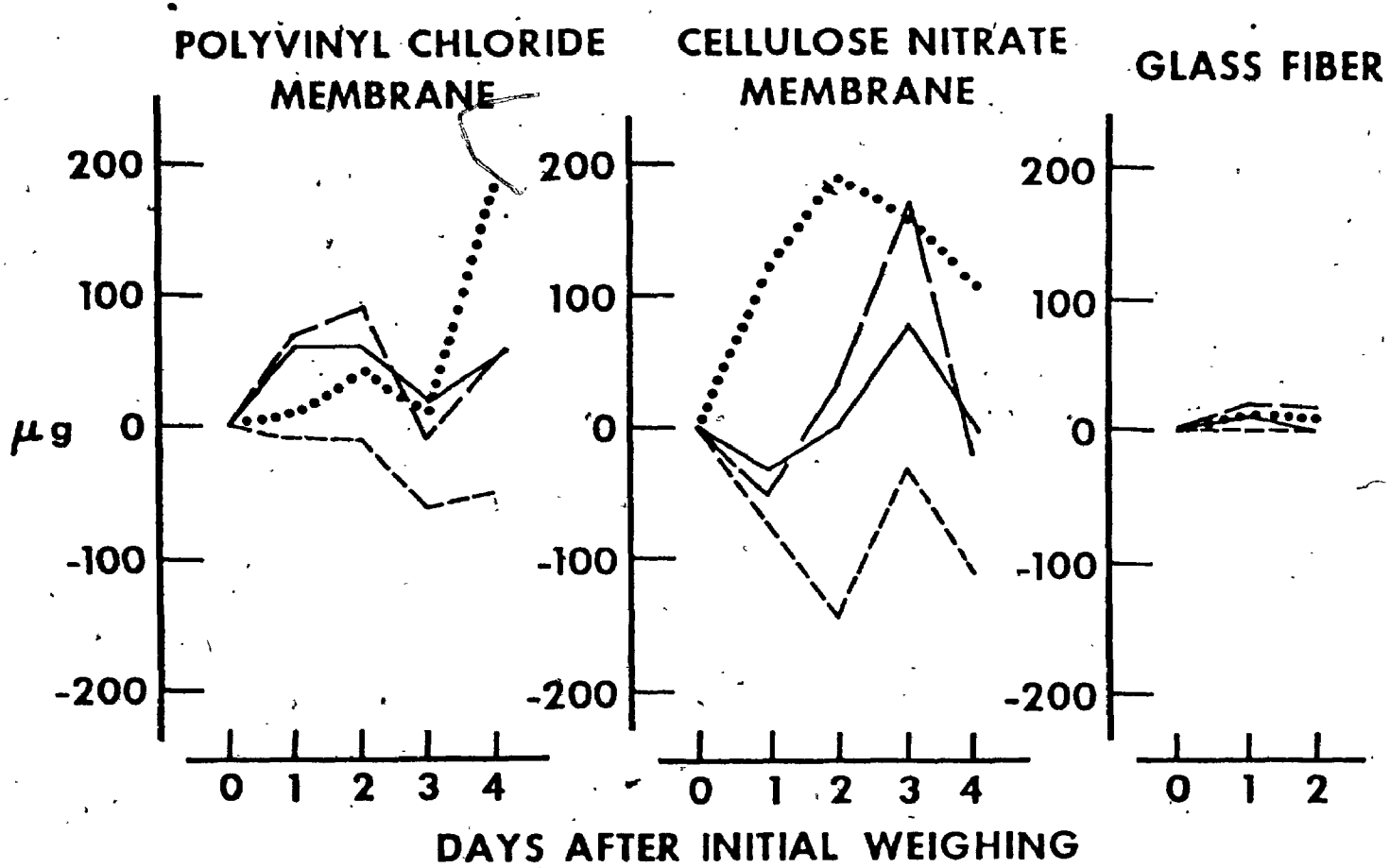
APPENDIX 5.2

Intersampler Correlation Lines for NO_2



APPENDIX 5.3

Intersampler Correlation Lines for RSP



APPENDIX 5.4

Weight Changes at Twenty-Four Hour Intervals of PVC, CN and GF Filters

APPENDIX 6.1

Percentage Prevalence Data of White, Non-Occupationally Exposed Non-Smoking Males and Females by Age and Area of Residence

AGE GROUP		7-14		15-24		25-44		45-64		TOTAL	
Residence		Leb	Ans	Leb	Ans	Leb	Ans	Leb	Ans	Leb	Ans
FEMALES:											
Usual Cough	%	9	7	3	12	8	18	13	19	8	15
Usual Phlegm	%	6	4	3	9	5	9	5	17	4	11
Ever Wheeze	%	11	11	9	12	17	12	20	26	14	17
Frequent Wheeze	%	2	0	3	4	5	0	5	9	4	5
Dyspnea	%	7	4	14	0	14	27	17	30	13	17
Total Tested		123	28	117	57	149	33	94	81	483	199
MALES:											
Usual Cough	%	6	0	3	10	9	11	15	8	7	9
Usual Phlegm	%	5	13	6	7	8	17	18	17	8	12
Ever Wheeze	%	16	13	14	7	19	17	9	8	15	11
Frequent Wheeze	%	0	13	2	3	4	11	3	0	2	6
Dyspnea	%	3	0	2	0	4	6	12	17	4	5
Total Tested		64	8	63	30	53	18	34	12	214	58

APPENDIX 6.2

DIFFERENCES BETWEEN MOST POLLUTED ANSONIA AREAS AND LEAST POLLUTED LEBANON AREAS

	MALES			FEMALES		
	N	CODE*	$rFEV_1$	N	CODE	$rFEV_1$
SMOKERS						
Ans MP	106	A	-.10	58	G	-.04
Leb LP1	88	B	-.21	58	H	-.11
Leb LP2	84	C	-.28	84	I	-.15
NON-SMOKERS						
Ans MP	33	D	-.09	100	J	.09
Leb LP1	25	E	.07	62	K	-.05
Leb LP2	22	F	.02	62	L	-.03

For Ansonia, population areas surrounding the most polluted (MP) sampling site (Site B) was chosen. For Lebanon, two sampling sites were of low pollution (LP1 = Site 1 and LP2 = Site 4) and the population areas around these sites were chosen.

* Codes were chosen for each category for statistical comparison. In males, there were no significant differences between groups for symptoms; for FEV_1, #C was significantly worse than #A ($P < .05$). In females, G and J had more cough than H and K ($P = .05$), but there were no differences in the other symptoms; for FEV_1, I was lower than A ($p = .05$) and K was lower than J ($P < .05$).

(Lebanon subjects reported significantly more farming than Ansonia subjects.)

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